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THE OPTICS OF CONTACT LENSES

BY

A. G. BENNETT

THE subject indicated by the title of this paper ranges over a wide field, but only that part of it which is of significance to prescribers has been dealt with here. An attempt has been made to present the subject matter as simply as possible without detriment to precision. Gaussian methods in particular have been avoided wherever they appeared unnecessary. On the other hand, attention is drawn to some commonly neglected factors which are of importance in contact lens prescribing.

1.—Neutralising the cornea

The idea of contact lenses was apparently conceived by Sir John Herschel after reading Airy's account of the correction of his own astigmatism by means of a sphero-cylindrical lens.

Herschel, though admiring Airy's ingenuity, evidently thought this too indirect a way of overcoming what he termed "malconformations of the cornea" and suggested instead a "spherical capsule of glass" filled with animal jelly and applied to the cornea. Thus the contact lens was first conceived as a means of re-moulding the cornea, or, expressing it more accurately, of building it up into a truly spherical surface, the function of the glass shell being purely mechanical and not optical.

Completely to neutralise the anterior surface of the cornea would require a fluid medium of the same refractive index as the corneal substance, namely 1·376, whereas the refractive index of all the solutions at present in use is slightly lower, being about 1·336. Since, for a given radius, the power of a surface is proportional to the refractivity (refractive index minus unity) of the medium, it follows that the fluid neutralises only 336/376 or approximately nine-tenths of the anterior power of the cornea. It will hence be realised that the effect of corneal irregularities or deformities is not completely counteracted.

The extent to which a contact lens neutralises corneal astigmatism is not a simple matter to determine. If we accept the constants of Gullstrand's No. 1 Schematic Eye as a basis for discussion, the following picture emerges. The corneal radii are 7·7 and 6·8 mm. respectively, its centre thickness 0·5 mm. and its refractive index 1·376, the index of the aqueous humour being 1·336. From this it can be calculated that the anterior surface of the cornea has a power of +48·83D., and the posterior surface of the cornea a power of −5·88D., the ratio of these two powers being −8·3 : 1.

Now if the corneal astigmatism is ascribed to its anterior surface only, it would follow as before that the fluid meniscus will correct nine-tenths and leave one-tenth uncorrected. If, on the other hand, both surfaces of the cornea are assumed to be toroidal with their meridians of maximum curvature in similar orientation, then the astigmatism due to the posterior surface will annul a portion of that due to the anterior surface. Suppose, for example, that both surfaces of the cornea exhibit the same percentage difference between the two principal radii, then since the surface powers are in the ratio −8·3 : 1, it follows that the posterior surface will neutralise nearly one-eighth of the astigmatism created at the first surface. The fluid lens neutralising nine-tenths for its own part, there would thus be a very small degree of over-correction.

One further point is worthy of mention. In order to make some allowance for the posterior surface of the cornea, keratometers are calibrated for an empirical refractive index of 1·3375.

For example, the dioptric reading corresponding to a radius of 7·7 mm. is $\frac{337·5}{7·7}$ = +43·83D., whereas taking the true index of 1·376 the corresponding power is +48·83D.

As a result, the reading given by a keratometer represents, in actual fact, 337·5/376, or very nearly nine-tenths of the power of the anterior surface. Similarly, the "corneal astigmatism" as measured by the keratometer represents in reality nine-tenths of the astigmatism due to the front surface only. The keratometer is incapable of giving reliable information as to the total corneal astigmatism. To accept its reading involves the assumption—which may or may not be justified—that the posterior surface is a replica of the anterior, with its principal meridians identically orientated, and its principal radii both reduced to approximately nine-tenths of the corresponding radii of the anterior surface.

2.—The Effectivity relationship

The optical theory of contact lenses cannot be adequately expounded without reference to the principle of effectivity, which deals with the change in the dioptric vergence of a pencil of rays over a given length of its path. (The vergence L dioptres at a given point is defined as the reciprocal of the distance l metres from the given point to the origin or focus of the pencil.) In passing it may be recalled that the first detailed exposition of

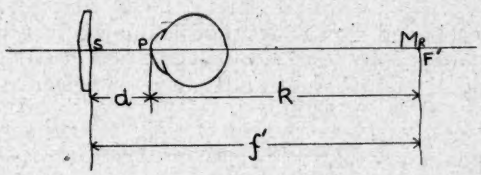

FIG. 1.

Spectacle and ocular refraction.

geometrical optics based on the idea of vergences was due to Sir John Herschel, who used the term "proximity" to denote the reciprocal of linear distance.

As an illustration we may take the relationship between spectacle and ocular refraction, which assumes some importance in contact lens prescribing. Fig. 1 represents a hypermetropic eye with its far point at Mr. In everyday practice, the refractive error is invariably expressed in terms of the correcting lens

which, placed at the " spectacle point " S, renders the eye emmetropic. The second principal focus F' of this lens must coincide with the far point. Its power F dioptres, the reciprocal of the focal length f' in metres, is termed the spectacle refraction. The distance in metres from the corneal vertex (more strictly, from the eye's first principal point) to the far point Mr is usually denoted by the symbol k and its reciprocal K is termed the ocular refraction. If the vertex distance from the spectacle point to the cornea is d metres, it is clear from the diagram that $k = f - d$ and hence

$$K = \frac{1}{k} = \frac{1}{f-d} = \frac{F}{1-dF} \quad \ldots \quad \ldots \quad \ldots \quad (1)$$

K represents the back vertex power of the contact lens required to correct an ametrope whose spectacle correction is F. By " contact lens " is meant, in this connection, the system comprising the glass or plastic shell and the liquid lens combined.

In general, a pencil with a vergence L dioptres at a given point will have the vergence L_d after travelling a distance d metres in air where

$$L_d = \frac{L}{1-dL} \quad \ldots \quad \ldots \quad \ldots \quad \ldots \quad (2)$$

If the pencil is travelling in a medium of refractive index n, d in this expression must be replaced by the " reduced distance " d/n. We shall need to make further use of this effectivity expression.

(The symbols and sign convention employed in this paper are those standardised by the Northampton Polytechnic Institute, London, E.C.1, and the Imperial College of Science.)

3.—Theory of afocal lenses

The first spherically ground lenses to be made available were produced by Carl Zeiss and were based on the Herschelian idea of reshaping the cornea. The glass lens itself was afocal in air. It is important to have a precise definition of this term; to say that it denotes a lens or system " without power " is not sufficient. An afocal lens is a lens without power with respect to infinitely distant objects; that is to say, a parallel pencil incident at the first surface emerges as a parallel pencil from the second. Such a lens does exert a focal effect with respect to near objects. That this distinction is not purely academic will emerge at a later stage.

Although the centre thickness of any contact lens is necessarily small, it is not so in comparison with the radii of curvature and, consequently, thin lens formulae cannot be used without introducing serious errors. Take, for example, a lens with a posterior corneal radius of 8 mm. and centre thickness 0·6 mm., the refractive index of the material being 1·5. If the front surface were also ground to a radius of exactly 8 mm. in accordance with thin lens theory, the resulting lens would be far from afocal: its back vertex power would be no less than +1·60D.

The precise relationship between the radii of curvature of an afocal lens is easily found. Denoting the radii by r_1 and r_2 respectively, the centre thickness of the lens by t and the refractive index of the material by n, the two surface powers F_1 and F_2 are given by the text-book formulae

$$F_1 = \frac{n-1}{r_1} \quad \ldots \quad \ldots \quad \ldots \quad \ldots \quad (3)$$

and $$F_2 = \frac{n-1}{-r_2} \quad \ldots \quad \ldots \quad \ldots \quad \ldots \quad (4)$$

r_1 and r_2 being in metres.

If we now imagine a parallel axial pencil incident at the front surface, its vergence after refraction will clearly be equal to F_1. After travelling a distance t metres towards its focus, its vergence will be increased to

$$\frac{F_1}{1 - \frac{t}{n} F_1}$$

in accordance with the effectivity formula. Hence, if the pencil is to emerge parallel, *i.e.*, with zero vergence, after refraction at the second surface, the power F_2 of the latter must be such that

$$\frac{F_1}{1 - \frac{t}{n} F_1} + F_2 = 0$$

whence $$F_1 + F_2 - \frac{t}{n} F_1 F_2 = 0 \quad \ldots \quad \ldots \quad \ldots \quad (5)$$

The above may be followed more easily by reference to Fig. 2, which indicates the vergence of the pencil before and after each refraction.

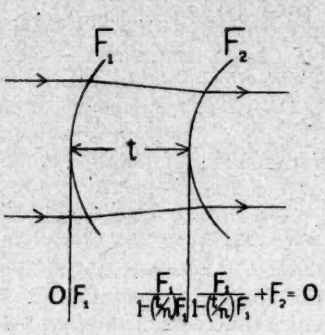

FIG. 2.

Passage of light through an afocal lens. The vergences are shown before and after each refraction.

The left-hand side of the last equation will be recognised as the formula for the equivalent power of a thick lens. The equation can be re-arranged in the form

$$F_1 = \frac{-F_2}{1 - \frac{t}{n}F_2} \quad \ldots \quad \ldots \quad \ldots \quad (6)$$

showing the necessary relationship between the two surface powers of an afocal lens. To obtain the relationship in terms of radii, we replace F_1 by $\frac{n-1}{r_1}$ and F_2 by $\frac{n-1}{-r_2}$, as above, and thus arrive at the equation

$$\frac{n-1}{r_1} = \frac{\frac{(n-1)}{r_2}}{1 + \frac{t}{n} \cdot \frac{n-1}{r_2}}$$

which simplifies to

$$r_1 = r_2 + \left(\frac{n-1}{n}\right)t \quad \ldots \quad \ldots \quad \ldots \quad (7)$$

Hence the radius of curvature of the front surface of an afocal lens must always exceed that of the rear surface by $\left(\frac{n-1}{n}\right)t$.

It can similarly be shown that in order to produce a contact lens with a given back vertex power, the required value of r_1 can

be found by adding $\left(\frac{n-1}{n}\right) t$ to the value yielded by thin lens theory.

The correction of ametropia with an afocal contact lens is entirely dependent upon the fluid meniscus, which is bounded by the posterior corneal surface of the contact lens (radius r_2) and the anterior surface of the cornea itself (radius r_c). It hence becomes necessary to determine the value of r_2 which will impart the required power to the fluid lens. An approximation to this value may be found as follows.

Considering the liquid lens as a separate entity and ignoring its centre thickness for the sake of simplicity, its power F is given by the thin lens formula

$$F = 1000\ (n-1)\ \left\{\frac{1}{r_2} - \frac{1}{r_c}\right\}$$

r_2 and r_c being expressed in millimetres. Assigning to n the usual refractive index of 1·336 this expression becomes

$$F = \frac{336}{r_2} - \frac{336}{r_c}$$

Since the liquid lens is in contact with the cornea, its power F must be made equal to the ocular refraction K. Hence we may write

$$K = \frac{336}{r_2} - \frac{336}{r_c}$$

$$\text{or} \qquad \frac{336}{r_2} = K + \frac{336}{r_c} \qquad \ldots \quad \ldots \quad \ldots \qquad (8)$$

Having calculated K from the spectacle refraction and measured the corneal radius on the keratometer, we can thus ascertain the required value of r_2.

Assuming, however, that the keratometer has its dioptric scale calibrated for a refractive index of 1·3375 in accordance with standard practice, the power C of the cornea as recorded on the instrument will be equal to $337{\cdot}5/r_c$ and hence we may substitute C for $336/r_c$ in equation (8) without introducing serious error. We thus obtain the following simplified expression

$$r_2 = \frac{336}{K + C} \qquad \ldots \quad \ldots \quad \ldots \quad \ldots \qquad (9)$$

The literature of contact lenses contains various graphical representations of this formula.

It should be pointed out that formula (9) is only approximate because it does not take into account the thickness of the liquid lens. This point would be worth further discussion were it not for the fact that the fitting of afocal lenses is no longer a commonly practised technique.

Its abandonment was largely due to the resulting conflict between optics and haptics. It can readily be calculated from formula (9), taking an average value of +43D. for the power of the cornea, that the correction of ocular ametropia over the range +12D. to −12D. requires afocal lenses with inner corneal radii varying from 6·1 to 10·8 mm. Indeed, at one time Zeiss afocal lenses were available with radii 5 to 11 mm. in half-millimetre stages. When it is realised that a lens with a 5 mm. corneal radius has a maximum corneal aperture of 10 mm., whilst a lens with 11 mm. radius cannot possibly clear an average cornea, one wonders how such lenses were ever made with any hope of a successful fitting.

It may be added that there is no virtue whatever in making contact lenses afocal and relying entirely upon the liquid lens for the correction of ametropia. From an optical standpoint it is quite immaterial how the total refractive power is divided between the lens itself and the fluid meniscus, whilst from the manufacturing standpoint a perfect afocal lens is certainly no easier to make than any other. On the contrary, small errors in power and defects of surface figure, waviness and so on, are more easily detected in an afocal lens than in one incorporating a refractive effect.

4.—Lenses with added power

Owing to the difficulties briefly alluded to above, it is now the usual practice to select the shallowest standard curve that will clear the cornea and to incorporate any additional power that may be required in the lens itself. Since even small variations in the fluid thickness may have an appreciable focal effect, it is important that refraction should be carried out with the patient wearing a contact lens identical in regard to its back surface and overall size with the lens that is finally to be worn. If any alterations are made, an attempt should be made to estimate their effect on the thickness of the fluid meniscus so that an appropriate allowance can be calculated.

The optical problem is illustrated in Fig. 3. The patient is presumed to be wearing either a semi-finished moulded lens or else a standard trial lens of the appropriate specification, and is found to require an additional power D at a distance d from

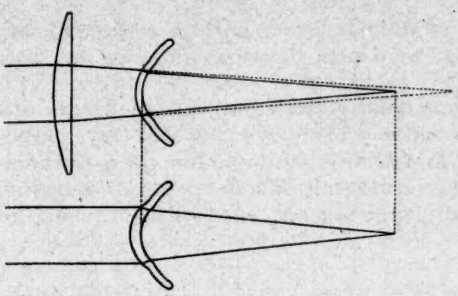

FIG. 3.

Basic optical requirement. The finished lens must have the same back vertex power as the trial contact lens and auxiliary spectacle correction.

the vertex of the contact lens. If the back surface of the latter is identical with that of the lens to be finally worn, the fluid meniscus will be the same in each case. It follows, therefore, that the back vertex power in air of the final lens must be made to coincide with that of the original system in air if it is to have the same effective power at the cornea.

The back vertex power F'_v of the system can be calculated as follows. Suppose the power of the auxiliary lens is +10D., the vertex distance d 12 mm., and that the contact lens worn during refraction is afocal with a posterior corneal radius of 8 mm., centre thickness 0·6 mm. and refractive index 1·5. According to formula (7) the corneal radius r_1 of the front surface is given by

$$r_1 = r_2 + \left(\frac{n-1}{r}\right)t = 8{\cdot}00 + \left(\frac{0{\cdot}5}{1{\cdot}5}\right)0{\cdot}6 = 8{\cdot}20 \text{ mm.}$$

Substituting in (3) and (4), the two surface powers are therefore 500/8·20 and 500/−8·00, giving

$$F_1 = +60{\cdot}98\text{D. and } F_2 = -62{\cdot}50\text{D.}$$

We can now find the back vertex power of the system by tracing through it a parallel pencil of rays incident on the auxiliary lens. After refraction by this lens, the pencil emerges with a vergence of +10D. and travels 12 mm. or ·012 metres in air before meeting A_1, the front vertex of the contact lens. In accordance with the effectivity formula (2), the increased vergence of the pencil at A_1 will be

$$\frac{+10}{1-{\cdot}012\times10} = \frac{+10}{0{\cdot}88} = +11{\cdot}36 \text{ D.}$$

Thus the 12 mm. air space has increased the effective power of the trial lens by +1·36D. However, as pointed out above, an

afocal lens is without power only in respect to infinitely distant objects and we must therefore proceed to trace the pencil through it.

After refraction by the front surface of the afocal lens, the vergence becomes $+11{\cdot}36 + F_1 = +11{\cdot}36 + 60{\cdot}98 = +72{\cdot}34$D. The pencil now travels 0·6 mm. though a medium of refractive index 1·5 before meeting A_2, the back vertex of the contact lens. Hence, again applying the effectivity formula, the vergence at A_2 is

$$\frac{+\ 72{\cdot}34}{1 - \dfrac{{\cdot}0006 \times 72{\cdot}34}{1{\cdot}5}} = +\ 74{\cdot}50\ \text{D.}$$

Finally, after refraction by the back surface, the pencil emerges with a vergence of $+74{\cdot}50 + F_2 = +74{\cdot}50 - 62{\cdot}50 = +12{\cdot}00$D. This gives us the back vertex power of the system, and the final contact lens to be worn permanently must be made with the same back vertex power in air.

Reference should be made to Fig. 4 (not drawn to scale) in which the vergences of the pencil are shown before and after each refraction.

Repeating the procedure for an auxiliary lens of power −10D, with all other particulars as before (see Fig. 5), it will be found

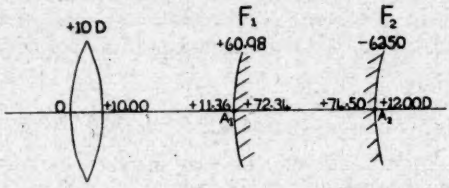

FIG. 4.

Computation of back vertex power: convergent auxiliary lens and afocal contact lens.

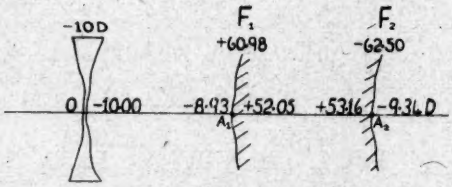

FIG. 5.

Computation of back vertex power: divergent auxiliary lens and afocal contact lens.

that the vergence at A_1 is −8·93D., whereas the back vertex power of the system is −9·34D.

Analysing these results we may conclude that the total effectivity correction consists of two portions, one due to the air space or vertex distance and the other due to refraction by the trial contact lens. When the auxiliary lens is of positive power, the two effects are additive; with a negative auxiliary lens they are in opposition.

In the above examples the contact lens worn during refraction has been assumed to be afocal, but exactly the same procedure should be followed in principle whatever its power.

Though difficult mechanically it is theoretically quite simple to incorporate an astigmatic correction in a glass or plastic contact lens. For technical reasons it is usually more convenient to make the inner corneal surface toroidal, in which case allowance must be made for the fact that when filled with fluid the back surface of the lens is partly neutralized. If the refractive indices of the lens material and fluid are n and 1·336 respectively, each dioptre of effective cylinder power will need $(n - 1)/(n - 1{\cdot}366)$, or approximately 3 dioptres of cylinder power worked on the lens itself.

5.—Magnification of the retinal image

An exhaustive investigation of this subject on orthodox Gaussian lines has been carried out by J. Boeder.[1] In his paper he deduced accurate formulae which took into account the effects of lens and fluid thicknesses. Unfortunately, one of Boeder's graphs seems to have been drawn upon by other writers, who have failed to study the accompanying text with sufficient care. As a result, a statement is sometimes seen to the effect that in myopia of 20D. contact lenses give a magnification of 46 per cent. In the sense in which it would be ordinarily understood, this statement is not even approximately true.

Supposing an eye to be corrected by a lens placed with its back vertex at a specified distance from the cornea, the size of the retinal image can be varied slightly by altering the form and thickness of the lens. This is, in fact, one of the expedients used in designing lenses for the correction of aniseikonia. Compared, however, with the change in image size introduced by placing the lens in contact with the eye instead of at the spectacle point, the effects due to form and thickness are of a secondary nature and can thus be neglected in a general discussion. In optical terminology, we shall assume the equivalent powers to be

[1] *Arch. of Ophthal.* (U.S.A.). January, 1938.

the same as the vertex powers. A very much simpler approach to the whole problem is thereby made possible and a Gaussian analysis can be avoided.

First of all we must establish certain definitions. Following Emsley[2] we use the term "Spectacle Magnification" to denote the ratio of the retinal image in the corrected ametropic eye to the blurred or sharp image in the uncorrected eye. With contact lenses it is easily shown (ignoring thicknesses) that the spectacle magnification is in all cases unity.

A graphical demonstration of this fact is given in Fig. 6, which represents a "reduced" hypermetropic eye, the corneal vertex being denoted by P and the macula by M'. Parallel rays are shown emanating from the extremity Q of a distant object situated

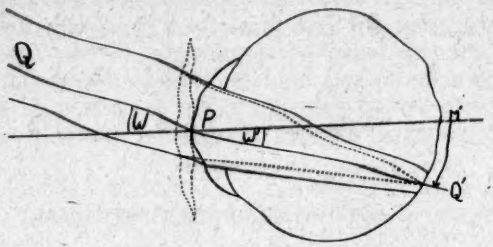

FIG. 6.

Spectacle magnification with a contact lens.

on the axis and making with it an angle w. The ray incident at P may be regarded as the central ray of the pencil entering the pupil. Hence, if this ray after refraction impinges on the retina at Q', then Q' represents the centre of the retinal blur circle. Furthermore, if the eye accommodates so as to neutralize the refractive error, the sharp image point formed on the retina will still be located at Q'. Thus in either case the size of the retinal image is M' Q'.

Suppose that the eye is now corrected by means of a thin convex lens (shown in dotted outline) placed in contact with the cornea. The central ray QP of the incident pencil passes through the optical centre of this lens and is thus undeviated by it, so that the retinal image point Q' occupies the same position as previously. In other words, the spectacle magnification is unity. The same argument obviously applies to the myopic eye.

The ratio of the retinal image size in the corrected ametropic

[2] "Visual Optics." Hatton Press.

eye to that in the schematic emmetropic eye is termed the "Relative Spectacle Magnification." For any given eye this ratio can be calculated only if the equivalent power of the eye is known. For this reason it is customary to develop two sets of formulae, one applicable to "axial ametropia" and the other to "refractive ametropia." This concept of ametropia as being either axial or refractive seems to the author altogether too schematic. It means, for example, that an eye hypermetropic 5D. must have a power of either +60D. ("axial" error) or else +55D. ("refractive" error)—no other values being admitted. Most of the published tables giving the "magnification" obtained with contact lenses are, in fact, tables of relative spectacle magnification on the assumption of purely axial ametropia. Although

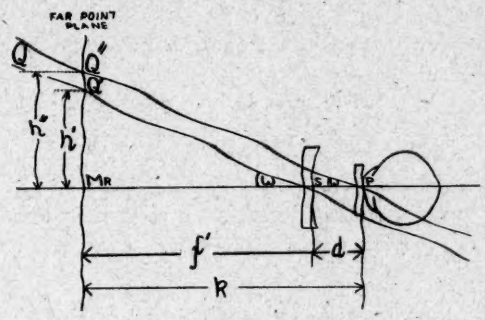

FIG. 7.

Comparison of image sizes in myopia between orthodox spectacles and contact lenses.

giving an indication of theoretical possibilities, their value in practice is questionable.

An exact comparison between orthodox spectacles and contact lenses can be deduced without making any such assumptions. To correct ametropia, a lens must form images of distant objects in the far point plane of the eye. Hence, if lens A forms an image in the far point plane 10 per cent. larger than lens B, the retinal images must also be in the same ratio irrespective of the power of the eye.

Fig. 7 illustrates a myopic eye with its far point at M_r corrected by a lens placed at the spectacle point S. Parallel rays are shown emanating from Q, the extremity of a distant object situated on the axis and making with it an angle w. Since rays passing through the optical centre of a lens are undeviated, the size h' of the image formed in the far point plane will be M_r

Q'. If the eye is now corrected by a contact lens, the size h″ of the image M_r Q″ formed in the far point plane is again determined by the ray passing through the optical centre. By similar triangles it will be apparent that the two image sizes are in the same ratio as the focal lengths of the lenses, *i.e.*,

$$\frac{h''}{h'} = \frac{k}{f'} = \frac{F}{K}$$

From formula (1),

$$K = \frac{F}{1-dF}$$

Hence

$$\frac{h''}{h'} = 1-dF \quad \ldots \quad \ldots \quad \ldots \quad (10)$$

Finally, putting d in millimetres, the percentage difference Δ is given by

$$\Delta = \frac{-dF}{10}\% \quad \ldots \quad \ldots \quad \ldots \quad (11)$$

Thus if F, the spectacle refraction, is −20D. and the vertex distance 12 mm., a contact lens will give a retinal image 24 per cent. larger than the orthodox spectacle correction.

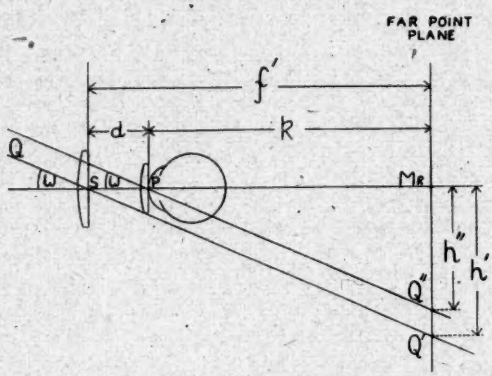

FIG. 8.

Comparison of image sizes in hypermetropia between orthodox spectacles and contact lenses.

Fig. 8 illustrates the position in hypermetropia, from which it will be seen that the same expression holds good. In this case

the smaller retinal image is given by the contact lens, the difference amounting to 7·2 per cent. in hypermetropia of 6D.

6.—Contact lenses in anisometropia

Assuming axial errors only, eyes with marked anisometropia will have different retinal image sizes, a fact which may by itself give rise to symptoms. Spectacles placed at the anterior focal points of the eyes would equalise the retinal images. Contact lenses would leave the disparity unchanged. On the other hand, contact lenses have the great advantage of obviating the unequal vertical prismatic effects arising with orthodox spectacles when the eyes rotate to view objects above or below the optical axis.

In unilateral aphakia, contact lenses have the advantage on both counts. The aphakic eye that was previously emmetropic or nearly so has a retinal image about 25 per cent. larger than its fellow, when the correction is worn at 12 mm. from the cornea. Contact lenses reduce this disparity to approximately 9 per cent. and it is stated that binocular fusion has thereby been rendered possible in certain cases.

7.—Contact lenses in near vision

Reference has been made above to the relationship between spectacle and ocular refraction. A similar relationship exists between spectacle and ocular accommodation, *i.e.*, between the nominal accommodation reckoned at the spectacle point and the actual effort of accommodation which the eye is required to make.

The left-hand side of Fig. 9 represents a hypermetropic eye corrected by a +6 D. lens placed 12 mm. from the cornea. Parallel rays after refraction by this lens reach the cornea with a vergence of +6·47 D. in accordance with the effectivity formula. In the lower

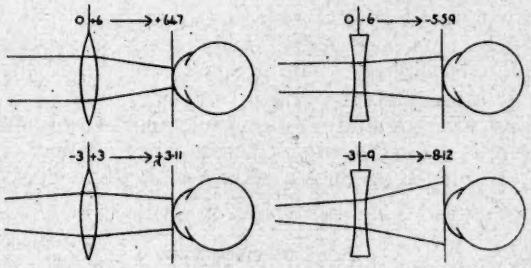

FIG. 9.

Spectacle and ocular accommodation.

half of the diagram, light is imagined to diverge from a point 33⅓ cms. or −3·00 D. from the lens. After refraction the vergence is +3·00 D., which is increased to +3·11 D. at the eye as shown. The effort of accommodation required to focus at 33⅓ cms. is therefore 6·47 − 3·11 or 3·36 D. Wearing a contact lens for distance, the accommodation needed to focus at 33⅓ cms. from the spectacle point or 34·5 cms. from the eye would be only 2·90 D.

The position in myopia is illustrated in the right-hand side of Fig. 9, which shows that a myope corrected by −6·00 D. at 12 mm. from the cornea has to exert 2·53 D. of accommodation in order to focus at 33⅓ cms. A greater effort of accommodation would be needed if a contact lens were worn.

8.—Sources of error and tolerances

We conclude with a very brief review of possible sources of error in prescribing and manufacturing. Assuming that the back vertex power required has been accurately computed as described above, there are still other factors to be considered. For example, an error of only 0·05 mm. in the inner corneal radius of the lens will produce an error in the refractive effect exceeding 0·25 D. Again, assuming the inner radius to be perfectly accurate, it requires an error of only 0·03 mm. in the outer corneal radius to alter the power by 0·25 D. A similar error in vertex power would result from making the lens 0·1 mm. too thick or too thin.

The thickness of the fluid meniscus is not a negligible factor, since every 0·1 mm. of fluid adds approximately 0·12 D. to the effective power of the contact lens system. An error in the corneal aperture will affect the thickness of fluid and thus alter the power. For example, an increase of 0·25 mm. in the corneal aperture will add about +0·12 D. to the power simply by increasing the fluid thickness. All the above figures are based on average radii and dimensions.

In short, there are several sources of error which may be small in themselves but may quite easily reach a formidable total if their effect is additive. The greatest care should therefore be taken by both manufacturer and prescriber.

One thing which a study of these tolerances has brought home to the author is the extraordinary performance required on the part of nature in order to produce a perfect emmetropic eye.

OBSERVATIONS ON THE PULSE AND RETINAL ARTERIAL PRESSURE

BY

Professor W. KAPUŚCIŃSKI

POZNAN

Part I—Arterial movements in the pulse

THE investigations into the pulse which I have the intention to make generally known by the present article, are the result of my studies of the problem of measurement of arterial movements in the central retina. Some 30 years ago these measurements were carried out for ophthalmological purposes by P. Bailliart, a French ophthalmologist, who in doing so made use of an instrument specially constructed by him and named by him an ophthalmodynamometer.

Bailliart's method found very wide application, and, in fact, the numbers of publications containing reference to results obtained by it, form to-day a veritable library. This method was the object of an individual theme on the programme of the last Congress of International Oculists in Cairo, 1937. I myself have devoted a good deal of time to this study.

The results of a number of different authors in relation to physiological arterial pressure are not in agreement, the deviation sometimes amounting to 100 per cent. It is therefore not at all surprising that someone has questioned: " What is the cause of this divergence, and is it due either to faulty technique or to an error in the method itself? " Desirous of solving this riddle, I began to inquire into the basis for our knowledge of the arteries. By adopting this procedure, I made reference to observations on the pulse, crystallizing in a separate whole anything relating to problems bearing on this truth. I therefore present in the first part of my article some facts bearing on the subject, although I am, at the same time, fully aware that I enter upon the sphere of the physiologist and specialist of diseases of the internal organs.

Shall I then be able to add something entirely new and nothing but " new facts " unknown to either physiologist or internist, to justify my action? What can I say beyond what physiologists and internists have proved experimentally and clinically as consolidated in text-books on physiology, and completing as such the theory on the pulse?

My reflections on the pulse are due to observations on the fundus oculi arrived at by a procedure on which neither physiologist nor internist has embarked so far.

What does it concern?

In the fundus oculi we see arteries and veins of a kind that we are unable to study from records anywhere. We behold them magnified 14 times.

On examining the fundus oculi, there can be observed the phenomenon of an arterial system, none too distinctly, however, as is often the case, and to this phenomenon little attention has been paid so far, and—it seems to me—that no explanation has ever been attempted.

This phenomenon sets in rhythmic motion the artery branching off from the central retinal artery. It is a movement which is sometimes very distinct, and sometimes most difficult to perceive. In a great many instances it is not perceptible at all.

The movement is to be seen most accurately in the bends of this artery, and the greater the bend, the more distinct becomes the motion.

FIG. 1.

On the above sketch I have endeavoured to explain this movement. We notice here two prominent peaks of the artery running in an inward direction, *viz.*, the peak *a* and the peak *b*. Simultaneously with the cardiac systole, the peak *a* moves upward to a point a^1 and the peak *b* downwards to a point b^1. Due to cardiac diastole, the peaks then return to their former positions *a* and *b*. The systolic movement is rapid, whereas the diastolic movement is slower. A diagram would present the following broken line:—

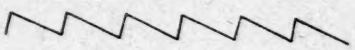

FIG. 2.

This broken line shows the pulse in angular outline, its first phase being rapid, and its second phase slow.

Between the peak *a* and the peak *b* there occurs a small interval where movement is entirely absent, let us call it the point of rest *c*. In the proximity of this point of rest, therefore, the artery performs movements which in the sections *a* and *b* differ from

each other. The smaller the bend of the artery, the shorter will be the rest, but the artery will always indicate two sections representing movements not in harmony with each other in the proximity of the point of rest.

It is very seldom that this rest is to be distinguished immediately on the flowing artery, but even then we are able to distinguish two sections characterized by a different flow near the point of rest in question, and we shall always be able to perceive a rapid systolic phase and a slow diastolic phase. In the proximity of the point of rest, the movement disappears completely.

According to Fig. 1, therefore, this movement takes place through *a* and *b* with the point of rest *c*, then continues through *e* and *f* with the point of rest *g*, and ceases completely when passing on through *h*.

This therefore represents the arterial movement in the fundus oculi in cardiac systole and diastole.

Here it is necessary to emphasize that no other movements are performed by the artery, that is to say, there are no other rhythmic movements in it to record, or, in other words, the artery neither dilates nor contracts.

I wish to point out once more that the movements herein described do not take place *distinctly* in all eyes. Let it suffice to emphasize, however, that they happen very often (in about 80 per cent. of cases), and that they are perceptible only by the application of a certain amount of skill. Their detection is possible when carrying out a series of eye tests for a considerable time in complete absence of movement, which, of course, is not within the reach of everybody. The investigation must only apply to the arch of an inward bent artery. The rhythm of the movements corresponds entirely to the rhythm of the pulse.

This phenomenon was by no means unknown long ago. Attention was paid to the views expressed on circulatory disturbances in Heine's "Augenuntersuchung bei Allgemeinerkrankungen" (Gustav Fischer, Jena, 1924). The opinion given therein is, however, quite erroneous, as I will expound later.

Among the literature to which I have had access, I have discovered only one publication by Ballantyne, the British ophthalmologist, who made a remark on the movements referred to above, confirming that they occurred in 36 per cent. of cases of eyes examined.

The nature of such movements had not until then been the object of special investigation.

In the meantime, the analysis of such movements has had an exceedingly far-reaching significance, due to Bailliart's method and to facts connected with the pulse in general.

Before proceeding to deal with the factors which are the direct cause of the arterial movement in the pulse, I am anxious to take into consideration the question bearing on my own observation, namely, *whether the arterial movements noticed in the eye are peculiar only to the central retinal artery, or whether this phenomenon can also occur in other visible arteries.*

In addition to the arteries of the fundus oculi, there are not many visible arteries. They are not seen *directly* as in the eye, but *indirectly* through the skin.

Under different circumstances, and in different places, arteries become visible in people. An artery, for instance, is ramified very differently in old persons of the emaciated and of the flabby type with high blood pressure. Thus the pulse of such an artery in the vicinity of the elbow joint, due to the running of collateral arteries, appears to be very different. They flow at a depth of 1 cm. or more under the skin.

We therefore observe the same movement as in the artery of the fundus oculi. But even in the other arteries we are able to perceive the pulse. In my personal self, for instance, these movements appear in the extension of the radial artery between the thumb and the index finger on the inner side of the left hand in its inward flow. It is also to be perceived by slight pressure with the palm on the ulnar artery in suitable illumination. These movements also appear in the temporal artery and in the dorsalis pedis. Further, it is seen in the femoral artery under Poupart's ligament. A certain amount of practice is required in order to be able to see it. Undoubtedly, there occur movements in the whole arterial system. *These movements are nothing else than the pulse.* This pulse, however, as it is felt in the radial artery, we interpret as rhythmic rising and falling of the arterial wall. This interpretation is erroneous, because it is obvious that the artery does not undergo expansion even to the extent of a fraction of a millimetre. The cause of this erroneous interpretation is the fact that the said artery runs in a groove between an offshoot of the radius and muscular tendons.

In this groove the artery can only perform its upward movement, and, therefore, the movement in question at the side of the finger—taken as an example—is such that we receive an erroneous sensation of the rhythmic dilatation and contraction of the artery. In all arteries where we feel the pulse, it is nothing but an arterial pressure movement and by no means dilatation of the arterial tube; *the arterial tube in cardiac systole and diastole remains unchanged.*

The above results in regard to the arterial movements are based on indubitable facts. These facts do not require any further discussion.

From these facts it is now necessary to draw adequate conclusions. Before proceeding with this, I will answer the question which is bound to suggest itself to everybody, namely: Are the above mentioned remarks known in literature, or has nothing so far been written on such obvious phenomena? It is here necessary to establish whether these facts are fully mentioned in text-books on Physiology. Neither in Beck's manual, nor in Tigerstedt's, nor Höber's (1930), nor Rein's latest edition (1943), nor Landois-Rosemann's (1944), nor Tigerstedt's fundamental 3-volume work on the revolutions in physiology, is there the least mention of these phenomena. Only in Beth's 1925 text-book ("Handbuch der normalen und pathologischen Physiologie," Julius Springer, Berlin), comprising about a dozen volumes, we meet in Vol. 7 in the section on the Arterial and Capillary Pulse by Frey on p. 1224 with the following remark: ". . . . . what we find in regard to the pulse is a change, as it were, in the position rather than in the volume of the artery during pulsation . . . ."

From this observation the author did not, however, draw any conclusion, because he did not fully estimate the importance of the phenomenon, and he contented himself only with the above cursory, suggestive remark. However little this writer, in fact, commented on this observation, there is a further remark in connection with his deductions on p. 1238, where he states: "under normal conditions this *vascular dilatation** at the time of cardiac systole is the measurement of the increase of pressure."

Thus the author reverts once more to the conception of vascular dilatation in systole.

Apart from the above brief remark, we cannot find any further reference to arterial pressure movements in text-books on physiology.

As regards French and English text-books, I have not been in the position of availing myself of these.

This negative evidence, however, did not satisfy me, for the very reason that it was hard to believe that the state of things described by me had altogether escaped the notice of scientists. I therefore set about my research work to find out the actual position of the question. To start with, this was a difficult task under the present circumstances. Thanks to the kindness of the junior colleagues of Dr. A. Horst, I came across a book published by G. Hauffe in 1930 on the cardiac pulse and circulation (Lehmanns-Verlag, Munich). It is a book in which the author struggles with his huge original treatises on the heart, blood stream, electrocardiogram, pulse, etc. The greater part of Hauffe's works is of interest outside my own sphere. In his

* The italics are mine.

observation, however, on arterial movements as described by me, he is in agreement on almost every point.

Moreover, Hauffe quotes earlier literature in which occur descriptions of the very same ideas. He quotes Volkmann who, as far back as 1850, wrote as follows: " If we lay bare an artery, we shall find that it assumes a tortuous position, and that it afterwards returns to a straight position " and " . . . . . measured by compasses, the arteries do not show a tendency to dilatation at the time of the pulse." Volkmann further cites Rudolphi, the predecessor of Johannes Müller in the Chair of Physiology in Berlin. Rudolphi proves that the arteries at the time of the pulse do not dilate. Hauffe also quotes Mackenzie who in 1904 wrote as follows: " What we perceive as a movement of the pulse, upsets our notion completely. By investigating this more closely, we shall find that the artery changes its position. A pulsating artery never undergoes a change in volume, but changes its position. The opinion that the pulse depends on the dilation of the blood vessel in question, or that it is due to the " throbbing " of its wall, is erroneous." I am quoting these authors according to Hauffe, but have had no opportunity of reading their works in the original. However, not a single one among these authors has offered any further suggestions with regard to the mechanism of the pulse on the basis of their observations.

Hauffe himself barely does it, and yet—all his arguments are not convincing. For example, Hauffe maintains that only the pulsating artery in the proximity of the place of pulsation assumes a curved course, either artificially or physiologically. This assertion is not quite correct. The arterial winding is unnecessary for giving rise to these movements, but the conclusive point is that the circulatory system be closed. In the arteries of the fundus oculi there occur no windings in spite of the pulse. This pulse has been the object of my present deductions. Thus Hauffe is unaware of anything concerning the arterial pulse of the fundus oculi.

Anyhow, after passing a review of the value of Hauffe's work, we have no doubt that he has expressed an adequate remark on exactly the phenomenon described by myself, though, of course, he has not drawn any serviceable conclusions at all from his observation.

I will now revert to the discussion of the true significance of the problem, namely, in relation to Bailliart's method. The question arises whether Bailliart arrives at the conclusion that there are indications of arterial movements in the pulse or, as the case may be, of any factors to which these movements are due.

As I have stated on the strength of my observations, movements

appear simultaneously with the cardiac systole and diastole. There can be no doubt whatsoever as to their being dependent on systole and diastole. Simultaneously with the systole, there results an arterial movement, and due to diastole the artery returns to its former position. As systole and diastole are factors prompting changes in pressure in the blood stream, the visible arterial movements are also exponents of such changes in pressure. If in systole and diastole the blood pressure remained unchanged (as maintained by Hauffe), these movements would not be due to this. The pressure would remain unchanged, if the arterial system remained an open system. (There would then occur changes in kinetic energy in systole and diastole, and also changes in the rapidity of the flow of the blood stream.) Turning it round the other way, the greater the difference between systolic and diastolic pressure, the more prominent would be the pressure movements.

We are, indeed, able to observe these changes in the fundus oculi. As I have already previously pointed out, these movements are hardly perceptible under normal conditions, and are often altogether invisible. On the other hand, in cases of rapid pulse, where the difference between systolic and diastolic pressure is known, for example, where it reaches 100, the arterial movements in the fundus oculi are very distinct, being visible without difficulty even to the naked eye. The conclusions mentioned a little while ago would immediately suggest to us that a difference between arterial systole and diastole of 0·1 mm. (arteries in the fundus oculi) is the minimum under normal conditions. This assertion is of immense importance for the estimation of Bailliart's method, but I do not now wish to dwell on it, and will therefore leave these arguments for a later part of my article.

In fact, I might as well deal with the pulse, as, after all, my present conclusions afford me a basis on which to develop my criticism of Bailliart's method. It is my intention, however, to solve the problem of the pulse as raised by me and to present it as a synthetic whole. I am of opinion that by doing so I shall be able to fill a certain gap in the theory of the pulse.

With this task in front of me, I must proceed from certain fundamental facts to arrive at a basis for my arguments.

I will try, first of all, to give an explanation of the origin of the arterial pressure movements, which gave rise to my inquiry. The proof that these movements have their origin in direct dependence on systolic and diastolic pressure does not yet explain the *mechanism* of the origin of these movements. To explain this mechanism, we must first confirm the fact that the circulation of the blood takes place in a closed system. As a matter of fact, any area of capillary vessels is several times larger than the aortic

area; this, however, the blood stream encounters in proportion as it passes into circulation, causing, when resistance is offered, a compression of the blood vessels and, above all, an increase of the viscosity of the blood. This kind of resistance determines the fact that the blood stream must be regarded as a closed system. At the same time, due to the cardiac systole, the blood receives the given impetus. This impetus produces an increase of pressure, which, according to Pascal's law, is distributed simultaneously throughout the arterial system. An impetus, taking the place of the "arterial bend," prompts arterial movement in the direction in which it works. The impetus makes itself strongly felt at the beginning of the circulation of blood, but, due to a number of factors, such as vascular contraction, viscosity of the blood, etc., this impetus weakens, and the kinetic energy changes into thermal energy. For the very same reason, the arterial movements also gradually decline in extent. In my opinion, this may also conveniently represent the mechanism of the arterial movements in the pulse.

As quoted by me, Hauffe compared the movement of the arterial system with the folding of a taut string. Such a comparison is not quite adequate, as in the case of the folding of the string both phases are equal. In fact, as I have already emphasized repeatedly, the phases are unequal, one being rapid and the other slow, a circumstance known to us from the pulse curve.

However, the problem of the pulse is not exhausted by the interpretation of the mechanism of resistance in arterial movements. This proof is of particular significance for investigations into retinal arterial pressure, but cover only a small section of the entire problem of the pulse.

The presence of arterial pressure movements as well as the lack of any traces of contraction and dilatation of the arteries in the pulse serve to establish facts which are in no way compatible with the present position of the theory on the pulse.

Allow me to enumerate the factors which, according to the present theories of the physiologists on the mechanism of the blood stream, contribute in playing a rôle.

The main factor is the cardiac muscle, acting as a compressing and suction pump. The assistant factors are the arteries. The work of the arteries, as already stated, is accomplished—according to the description in all text-books by physiologists—as follows: The cardiac systole causes the blood wave to be thrust onward to the artery, and dilates it in a certain limited section; in the next phase, due to the action of the muscle, the artery contracts. This arterial movement takes place throughout the

arterial system, giving rise to the same kind of peristalsis. Due to this arterial movement, therefore, there is, on the one hand, a perceptible pulse and, on the other hand, peristaltic movements serving the purpose of a powerful factor in setting the blood stream in circulation.

In contrast to this theory, we have in Beth's treatise the views on vascular peristalsis.

In the chapter on the auxiliary function of the blood vessels in the circulation on p. 1071, its author, Alfred Fleisch, while basing himself on his own evidence and that of Hürthl, arrives at the conclusion that arterial peristalsis does not exist. To quote his own words, Fleisch writes on p. 1083 as follows: " . . . by this assertion it is confirmed that neither large nor small arteries perform any systolic movements. The arteries are provided with the very same tubes, but do not produce any active force to set the blood stream in motion." Fleisch also denies the existence of a so-called " cardiac circulation " having its say in the peristalsis of capillary vessels. It need not be emphasized that Fleisch's views in their essence are in accordance with my observations.*

These deductions on the part of Fleisch have not found their way to text-books on physiology, so that the views expressed in the foregoing still prevail in the science of to-day.

The theory on the pulse denies the facts suggested by me: the pulsating artery (with the exception, perhaps, of the commencement of the aorta) neither dilates nor effects a peristaltic movement.

Let us, however, attempt once more to consider critically this theory on the pulse dominating as it does all text-books on physiology. According to this theory, the work of the cardiac muscle would not be made use of in the onward motion of the blood stream in the circulation, but a certain part of this force would be used in surmounting the resistance of the vascular muscle and this, in fact, simultaneously throughout the arterial system. This simultaneous dilatation of the arterial tubes of the whole circulatory system would necessarily cause reduced pressure in the whole system.

* With regard to the theory on the pulse expounded in the above mentioned text-book, one cannot help noticing that there exists an appreciable divergence of opinion which, I venture to say, leads to utter confusion. Two authors, Frey and Fleisch, have worked on the same subject, advancing views that are altogether different. Undoubtedly, Fleisch arrives nearer the truth, though even he comes to a standstill, as it were, in the pursuit of his investigations. He contents himself by denying the co-operation of the vessels in the onward thrust of the blood stream. This denial is based on the lack of peristalsis in the artery. However, the opinion of Fleisch is only partly correct. The lack of peristaltic movements does not so far prove that the artery has an active share in the onward thrust of the blood stream. A little further on, however, he states that the arteries prove to be in apparent co-operation in the onward thrust of the blood stream, though they do not perform peristaltic movements.

As regards the following (false) arterial systole which would level out the pressure, and would thrust the blood onward in circulation, it is obvious that this could happen only if the systole is peristaltic, as, indeed, taught by the physiologists, but there has been no one hitherto able to prove it.

The *cardiac* systole would correspond to the *arterial* diastole and, *vice versa*, the cardiac diastole to the arterial systole. When examining blood pressure, we distinguish between systolic and diastolic pressure. Systolic pressure we should still describe, in accordance with the views held up to now, as the phenomenon when the artery dilates due to cardiac systole, and diastolic pressure when it contracts.

In consideration of this, it follows that systolic pressure at the time of arterial dilatation must be lower than diastolic pressure at the time of contraction. This is obviously inadmissible, but according to the present views on the rise of the pulse, I do not see any possibility of grasping this matter otherwise. It is necessary also to remember that a hardened artery, sometimes hard as lead, pulsates more rapidly than a soft artery, and it pulsates not because of dilatation of its tube, but due to a pressure movement affecting the whole artery to be felt by our testing finger.

Thus we shall find that the theory hitherto on the pulse and the circulation of the blood cannot be criticized merely on the ground of this theoretical consideration. Yet, basing ourselves on arterial pressure movements, and in consideration of the non-occurrence of any apparent contractions or dilatations of the arteries, it proves absolutely untenable.

Further, it is necessary to prove that the third factor which, according to the text-books of physiologists, is an auxiliary force in the blood stream, in the sense given by physiologists does not exist. Such being the case, the question arises why the two factors connected with the cardiac action resembling a compressing and suction pump, are not sufficient for the blood stream.

Now we have also to prove that if the blood stream were dependent only on these two factors, it would not reach the perfection which indeed it does.

The German surgeon Bier expressed the following opinion: When performing the amputation of a foot, there arises the question of Esmarch's bandage. As soon as the amputation has been carried out, it is necessary to cover up carefully all arteries before the removal of the bandage in order to prevent haemorrhage. In cases of hardening of the arteries the bandage may be removed before tying up the vessels, and no blood from hardened vessels will flow out. I quote this statement on the responsibility of Hauffe, who, however, does not refer to it in his book.

What does this statement imply?

It simply implies that in non-hardened arteries the third factor, the motorial, operates where the second factor, the suctorial, is eliminated at the resection of the vessels. In hardened arteries, however, it does not function. The question also arises how the third factor is functioning and whether it is functioning in the way described in the text-books. In order to comprehend the working of that factor, let us realize its general significance from what is known from physics. The basis for the experiment selected is a row of elastic balls suspended on cords. The balls are placed so that they touch each other. We lift up the first ball, thereby giving it potential energy. We then let go this ball, so that it touches the remaining row of balls, when the last ball in the row rebounds from the rest and the others remain motionless. What do we witness here? The potential energy given to the first ball changes into kinetic energy during the lowering of that ball. This kinetic energy at the striking of the remaining balls changes into potential energy, which once more becomes kinetic energy in relation to the last ball when moved from its place.

The same thing takes place in the blood circulation, *viz.*: potential energy created by the systole of the cardiac muscle is transferred to a section of the artery as kinetic energy, thereby effecting arterial movement. At the same time, a part of that energy continues as potential energy at the wall of the artery, which immediately restores it to diastole in the form of kinetic energy of the blood stream. In the event of the vascular walls being hardened, the change of potential into kinetic energy becomes much reduced, owing to the third factor losing its force of action in a large measure. Such being the case—as already quoted by me from the text-book—the potential energy given to the blood by the heart diminishes gradually to a limit varying due to the effect of several factors connected with calorific energy, and, as a result of its centrifugal action, it does not hamper the circulation of the blood, but is in harmony with it.

As quoted by me above, Hauffe likewise attempts to explain the task of the third factor.

Thus Hauffe refers to the rubber tube test cited by him on a previous occasion. In physics this test is known as "Bernouille's test."

He bases his explanation on the following observation: If we admit through an open rubber tube a stream of water, the swifter the current of water, the tighter will become the tube. The same phenomenon can be seen on an X-Ray screen in the aorta. Hauffe proves that the artery contracts due to cardiac systole, and that

diastole causes it to dilate. Simultaneously with the dilatation there appears tension in the arterial wall, by way of potential energy, which at the following systolic movement is converted into kinetic energy assisting the onflow of the blood stream.

Hauffe was not aware of the publication of Bronislaw Sabat's article in the Lwow Medical Compendium in 1911. In this article Sabat describes the radiokinographical method employed by him, which he applied to all organs performing movements. By the aid of this method he was able to prove that the aorta, above all, performs pressure movements. Bernouille's phenomenon of the aorta does not arrive nearer the truth.*

I therefore think that the third factor, the acceptance of which seems indispensable, is performing its work in the way demonstrated by me.

In connection with the actual behaviour of the mechanism of the blood stream, there arises a series of problems† the mention of which would exceed the limits of my task, the more so as some of them would require experiments which under the present circumstances I am unable to carry out. Nevertheless, I should like to dwell on two problems connected with theories developed by me, although they happen to be outside the scope of my present subject.

I wish to bring further light on some remarks on the pulse. Above all, it is my desire to make it known once and for all that I have no intention to review the whole of this problem. In consideration of the immense work and the numerous experiments devoted on this problem, a full account would necessitate a separate monograph. Moreover, in order to give conclusive evidence in such a problem, it would be necessary to do so, in the first place, by way of proofs which, as stated above, I am unable to produce. I only intend to make a few remarks which—it would seem to me—have a bearing on my previous deductions.

As quoted by me above in a number of instances, Hauffe proves that the pulse is an artificial phenomenon due to arterial pressure. In my opinion, Hauffe's proof is unquestionably correct. In the absence of arterial pressure, the pulse curve would necessarily describe a different course, *i.e.*, the curve would not develop

* A description of the same method was published on the appearance of Sabat's article by Gött and Rosenthal. In a monograph on kinography Prof. Pleikart Stumpf makes mention of Sabat as the first to give a description of the radiokinographic method. After the appearance of Sabat's article, my attention was called, among others, to Prof. K. Mayer's remarks, for which I tender my heartiest thanks.

† In the physiology of the text-books there is quoted the rubber tube test by way of an example of the circulatory conditions and the appearance of the waves. In the rubber tube one is able to reproduce the peristaltic wave movement and to finish it off with the rebounding effect in question. On this comparison depends the unfortunate misunderstanding which led to the distortion of the theory on the pulse. However, a rubber tube device can be identified with an artery only with due reserve.

capriciously due to pressure, but would necessarily reflect any movement emanating from the artery. What the curve would look like then, I cannot tell. I do not think the completion of such evidence under favourable circumstances would be a difficult matter. Unfortunately, it is not possible for me to do so. It would be of uncommon interest, were it proved that such a curve appears also in a dicrotic phase.

As regards this dicrotic phase in the pressure pulse, it is difficult to become reconciled with even the best of theories. This phase must necessarily be simultaneous with the rebounding of the pulse wave from the capillary area. Such a theory, however, is not altogether acceptable. The pulse wave, for the reason that it fades out in the area in question, changes—as I have already mentioned in the foregoing—into thermal energy. Moreover, the pulse wave proceeding from the cardiac valve, cannot change, but—as we have proved—must flow onwards, and not backwards. Such a wave could reverse its course only in the event of a standstill of the blood pillar, but could not run against the current in the wrong direction.

Whence, therefore, does the dicrotic phase arise, and what is the origin of occasional protuberances that may occur?

Hauffe conjectures that they are an expression of the throbbing of the entire arterial system, a throbbing which continues from the first impetus due to the cardiac muscle systole. He compares this throbbing to the vibration of a taut string.

It is necessary to remember that the pulse wave can manifest itself only in the arterial walls. It then manifests itself as an arterial pressure movement produced by changes in the pressure of the blood stream; inside the artery in the vicinity of the arterial walls enclosing the liquid the blood stream indicates changes in pressure only periodically. A manometric test shows a curve corresponding to the pulse curve, because it also covers the dicrotic phase and other vibrations. In this curve obtained manometrically, as well as in the pulse curve, the continued vibrations cannot be produced by a return wave.

How is it possible to conjecture such a thing as that the blood stream should arrive at a complete standstill, as asserted by Franke, in order to flow back to the heart?

Is it possible to show a greater waste of energy of the heart, should this theory prove to be the true state of things? The heart has to spend its energy on the dilatation of the vascular tubes of the entire arterial system, and afterwards indirectly, due to peristaltic contraction of the dilated artery, has to serve the purpose of a permanent blood pillar impelled to supply the circulation. Instead of profiting by the existing blood stream, the

cardiac force and, indirectly, the arteries, must 72 times a minute move on the blood arriving at a standstill. Is it possible that some motor power or other disposes of that energy so uneconomically?

The blood stream cannot arrive at a standstill. From the first phase in the genesis of its existence when the blood serum is collected from the circulation in a cardiac tube, the heart is through this tube in direct communication with the arterial system. From this moment, the onflow of the blood stream continues uninterruptedly, receiving a constant rhythmic stimulus due to the cardiac systole. Thus it happens that an inert force is stirred to life, commencing with the first cardiac systole until death, and until the heart stops beating; the blood stream on death no longer receives an impetus, as there is no outflow from the arterial system, the arteries being left empty at death.

This brings my reflections on the pulse to an end, and I will now give the following brief summary. In the fundus oculi we observe simultaneously with the pulse, arterial movements depending on the impetus given by the local artery. These movements appear in two phases. The first phase, due to cardiac systole, is rapid, the second, due to diastole, slow. Any other movements, viz., arterial dilatation and contraction, do not take place. We observe the same pressure movements in all arteries of visible organisms, and there are no other movements performed by the arteries. The pressure movements in question are directly due to cardiac systole and diastole, and arise as the result of periodical changes in arterial pressure. Notwithstanding that the arteries do not perform peristaltic movements, they are, thanks to their structure, able to give an impetus to the onflow of the blood stream in the circulation on the basis of simple mechanical laws. The arterial pressure movements are actually the pulse. The pulse therefore does not depend on arterial dilatation and contraction. The above mentioned facts about arterial pressure movements together with the complete immobility of the arterial tubes, lead to the conviction of the unreality of the present theory on the cause of the pulse. A "false" pulse is met with in the artificial product caused by pressure. The blood stream in its first phase originates at the genesis of life, and continues to flow on uninterruptedly, receiving a rhythmic stimulus due to cardiac systole.

Part II – Measurement of pressure in the retinal artery system

I will now pass on to the discussion of Bailliart's method, and wish once more to emphasize the very great practical and theoretical significance to be ascribed to this method. In support of this

test there arose a number of theories, such as the theory of the rise of choked disk or papilloedema, pseudoneuritis of the optic nerve, and atrophy of the optic nerve in tabes dorsalis (locomotor ataxia) and in glaucoma (Sobanski, Lauber).

Various other optical disturbances and retinal troubles due to vascular disturbances, have been revealed, as it were, by means of the ophthalmodynamometer. The extent of interest attached to Bailliart's method in the ophthalmological world has of late years diminished considerably. As far as Schiötz's tonometer is concerned, it supplied an indispensable need in connection with the question of ocular instruments in cases where Bailliart's ophthalmodynamometer very often proved useless, as generally happens in the hands of practitioners.

It would seem accurate to say that the significance of the ophthalmodynamometer should in no way be regarded as less than that of the tonometer. The study of the conditions of the circulation of the blood in the retina must have a significance equal to the examination of blood pressure in the arterial ramification and, maybe, even surpass it in importance. The present investigation, however, claims the possibility of estimation not only of arterial pressure, but also of the main conditions in the venous system, resulting from the material relations of both systems, and also in relation to general blood pressure and internal pressure.

Such claims on the part of Bailliart's method are outside its scope, or come into consideration only on a very limited scale.

The most appropriate reproach which can be made against this method is the divergence of the results obtained thereby. The divergence is here very great indeed. I permit myself to quote the results of some authors in regard to arterial pressure:—

	Diatolic pressure	Systolic pressure	
Sobánski	48 — 56	80 — 90	
Bailliart	30 — 35	65 — 70	
Duverger Barre ...	50 — 60	80 — 100	Quoted from Weiss's "Kurzes Handbuch für Ophthalmologie."
Samoilow	35 —	80 — 85	
Magitot and Bailliart (in the dog)	50 — 65	80 — 90	
Blidung	70 —	117	

This divergence was lately emphasized by Behr ("*Zentralblatt für gemeine Ophthalmologie,*" *p. 70*). I do not intend to repeat these deductions, but would refer the reader to the article in question.

There are, however, other sides of the problem that are of paramount interest to me, and I am anxious to find out whether the divergence of the above-mentioned deductions depends on the technique employed in the investigations, or whether it is also due to the method itself. I should like to speak critically of the soundness of the basis of the whole method as such, and it is possible that the basis of this method must be questioned, and that therein is to be sought the fundamental error which is the cause of the deviations of the results arrived at (and, at the same time, their contradiction to established facts). The method of investigation of retinal arterial pressure, in fact, is borrowed from the method employed in the determination of pressure in an arterial ramification. In consideration that the method of investigation of pressure in an arterial ramification does not call for any fundamental reservations of being of direct advantage to the method itself and of ensuring exactness as to results, we will examine, one by one, the factors presented to us in the course of this evidence.

We apply on the forearm a bandage which must be sufficiently wide. By this bandage we exercise indirect pressure on the brachial artery. The amount of this pressure we measure by means of a suitable instrument. The factor is the pulse in the radial or ulnar artery. The moment when the pulse begins to decline is chosen for the measurement of diastolic pressure, and the moment when the pulse becomes incapable of being felt for that of systolic pressure.

What is then the method of measuring retinal arterial pressure according to Bailliart? First of all, as necessarily appertaining to the technique of the method, we examine the pressure in the brachial artery: Thus, in the first place, the artery must contract, as a fundamental condition, on a wide distance in the front of the eyeball and, at all events, in front of its ramification in the vascular funnel; secondly, the factor must be the pulse in one of the ramifications of the central artery, that is to say, the *spontaneous* pulse as met with in the above-mentioned artery. This is the way, therefore, the experiment has to be carried out to serve the purpose of an adequate method of measuring blood pressure in arterioles. We will now examine what actually happens when applying Bailliart's method to measurements of central retinal arterial pressure. Here, likewise, we exert indirect pressure on the artery, but not on the artery in the vascular funnel together with its ramifications. We

observe then the so-called arterial pulse. This "arterial pulse" depends on the following phenomenon: In connection with the pressure on the eyeball at any given moment we perceive rhythmic arterial contraction and, as the case may be, dilatation. The resulting arterial movement served Bailliart as a factor for determining arterial diastolic pressure in the central retina, and, on the other hand, as a factor for arterial systolic pressure, the decline of that movement at increased pressure on the eyeball. *The rhythmic arterial contraction and, as the case may be, dilatation of the central retinal artery is generally known to ophthalmologists as a spontaneous arterial pulse.* It is exactly in this restraining procedure that the first error of Bailliart's method is involved.

Thus this method was adopted on the suggestion of the view held hitherto on the essence of the pulse, it being believed that it depends on the rhythmic dilatation and contraction of the arterial tube. A similar phenomenon was noticed in the retinal artery, and these movements were regarded as sufficient to determine the pulse. The suggestion afforded by the theory held so far on the pulse was altogether too great, and yet, it had not even been ascertained whether the rhythm of the movements in the central artery is altogether different from that of the pulse. The theory on the pulse sets forth that due to cardiac systole there appears, first of all, arterial dilatation, and due to diastole-arterial contraction. The dilatation is a rapid phase, but the contraction a slow one. With reference to pressure "pulse," it is not ascertained whether the phases in the retinal artery happen in inverted order, so that, first of all, due to cardiac systole, there is a rapid phase expressing itself by way of *contraction*, followed by a slow phase, due to diastole, indicated by *dilatation*. The origin of this "false" pulse and of slow factors I will discuss in detail in the following.

For the present, I would emphasize that there is no analogy between the method of examining pressure in an arterial ramification and that used in determining pressure in a central retinal artery, there being only an apparent resemblance, due to a similarity of mechanism. When taking into consideration all the above-mentioned facts, it is necessary to prove that the difference between these two methods of determining pressure depends on the following points:—

First, on the change in pressure exerted directly on the eyeball, i.e., the organ in which arterial pressure has to be investigated.

Secondly, on the completely changed factor, because the "pulse," which we regard as the factor, has nothing in common with the spontaneous pulse due to pressure. To the spontaneous

pulse described in Part I of this article, attention has not been paid in full.

Thirdly, on the evidence of completely changed conditions in the organ examined, which are discovered when the examination is carried out under increased pressure.

It is, however, possible that these variations in the method of investigation are no fundamental errors, and that this method does not lead to false results. In other words, it may be questioned why, despite the changed conditions in the way of carrying out the examination, the results of Bailliart's method, nevertheless, do not give us an insight into the true conditions of pressure in the retinal arterial system.

Let us endeavour to examine this problem from its purely theoretical standpoint. According to Bailliart's method, the proportion between systolic and diastolic arterial pressure equals 0·45 : 1. However, such an estimate cannot be absolutely in correspondence with the true state of things. Due to the circumstance that in the retinal arteries the spontaneous pulse is not altogether apparent, but, in the majority of cases, a hardly perceptible or an entirely imperceptible feature, the difference, therefore, between systolic and diastolic pressure must be very much less. The reason for this assumption is that the pulse movements, as shown in Part I of this article, are dependent, above all, on the difference between systolic and diastolic pressure. If the relation between systolic and diastolic pressure was, in fact, equal to 0·45 : 1, then the margin would exceed that in the brachial artery, where the proportion is, more or less, 0·6 : 1.*

The physiological pulse in the retinal arteries should therefore be quite perceptible in every eye, and should even be more distinct than in the above-mentioned artery.

In consideration of the fact that, as already mentioned, the physiological pulse is much lower than would even appear to be the case, we must conclude that in the central retinal artery the difference between systolic and diastolic pressure is inappreciable. In diseases of the arterial system, in valvular trouble or, in general, in cases of rapid pulse, it is bound to be very much more distinct

* The manometric test made by Duke-Elder on cats, gave the following results: in the central artery 59/69 to 83/94; in the ophthalmic artery 53/85 to 108/129, corresponding to the proportion of 0·7 : 1 in the central artery, and the proportion of 0·5 : 1 or 0·66 : 1 in the ophthalmic artery. The difference therefore in the central artery between diastolic and systolic pressure is less than the difference shown by Bailliart's method (0·45 : 1). However, Duke-Elder's method depending as it does on the aperture due to inserting a glass tube into the arterial tube, must necessarily show this reduced value. It would, nevertheless, be necessary to prove that this method, the subtle technique of which must be admired, does not change the conditions of arterial pressure.

than in cases of natural flow of the blood stream, as already mentioned in Part I of my article. The foregoing theoretical considerations therefore lead to the suggestion that here occurs a fundamental error, due to Bailliart's method. There also occur other shortcomings, for example, the maximum pressure indicated by Bailliart's method is doubtful. The maximum systolic pressure amounts to—more or less—80 mm. Hg.

In view of the fact that the pressure in a vein must amount to, more or less, 20 mm. Hg, it would have to be proved that this great decrease in pressure occurring within the eyeball along a distance of 4 cm., can also be considerably less. The maximum reduction in pressure occurs in the arterioles. The maximum resistance in the circulation of the blood is created in the arterioles and capillary vessels, but physiology does not provide us with any accurate measurements. We are, however, able to assume that in the retinal arteries which count with fractions of a millimetre, the pressure is bound to be reduced considerably (as regards the pressure in capillary vessels to the extent of at least one-eleventh of the general blood pressure). We shall also find that the velocity of the blood stream in capillary vessels is exceedingly small, amounting to no more than a fraction of a millimetre per second. The interpretation of the appropriateness of this spontaneous flow is probably superfluous. For the maintenance of this spontaneous flow, the assumption must be made of a gentle and by no means sudden reduction in pressure. Moreover, one is bound to be struck by the non-correspondence of the reduction in pressure from the large blood vessels to the central retinal artery : 130 – 80 equals 50, and from the said artery to the vein : 80 – 20 equals 60. The above-mentioned theoretical considerations agree with results based on the true facts connected with the physiological pulse, thereby emphasizing further the fundamental error of Bailliart's method.

For the further elucidation of this interesting problem we will in the following deal with the origin of the " pressure pulse."

It is a most significant fact that ever since the dawn of the era of investigation, that is to say, for nearly a century, inquiries have been made into the origin of the venous pulse, and even to-day debates continue on that question. On the other hand, the manifestations of " pulsation " in the arteries are not heeded at present, and the interpretation given 90 years ago, as formulated by A. v. Graefe, is still accepted.

As a matter of fact, v. Graefe proves that at a moment when due to pressure on the eyeball the pressure within the eyeball is higher than the diastolic pressure of the central artery, the artery at that very moment must begin to pulsate, and, provided only that the

flow is accompanied by pulse waves, the arterial pressure will remain sufficiently high to enable blood to be pumped into the inner section of the artery.

Before making a critical investigation of the arterial " pulse " in the eyeball, we have to examine in brief the problem of the venous " pulse." It will not be necessary to mention in detail all theories expounded on this subject. Reference is only made to Serre's article in the *Arch. f. Ophthal.*, 1937, in which the author established the passivity of the venous pulse on the basis of cinematograph films, and it is impossible that it could be otherwise. The ocular veins cannot but " pulsate " passively. The type of arterial fluctuating pressure is experienced in the large veins in the vicinity of the heart, where the pressure is negative and where fluctuating pressure occurs (vestibule of vena cava). In the veins of the circulation the blood stream is uniformly without any fluctuation in the pressure. The retinal veins are by no means an exception. It is therefore out of the question that, as suggested by Türk and, in the main outlines, by Sobanski, the venous pulse is a spontaneous " pulse " caused by the transfer of the arterial pulse by way of capillary blood vessels to the veins. This pulse, however—I repeat this fact once more—is apt to decline in surroundings where the potential energy is transformed into caloric energy. It cannot therefore be denied that the arterial pulse is transferred by means of capillary vessels to the veins.

There cannot be any doubt whatsoever that the " pulsation " in the veins is a passive type of " pulsation " produced by fluctuating interior pressure. Simultaneously with cardiac systole there is propagated to the eyeball a wave of blood, which engages in rhythmic fluctuating interior ocular pressure. No other force can here come into consideration. The venous blood-pressure equals the lower pressure in the interior of the eyeball. It can be higher—even considerably higher—but can never be lower than the latter. *We can measure this pressure by Bailliart's method.* In this connection, I should like to point out from what has been stated above that by Bailliart's whole test method only in the investigation of venous blood-pressure do we arrive at results which are, more or less, in accordance with the true state of affairs, but one thing is quite clear, and that is that this method is not serviceable in measuring systolic and diastolic venous blood-pressure, for, as I have pointed out in the foregoing, such a difference does not exist at all. The possibility of an accurate definition of pressure seldom presents itself, and this for the following reasons: the height of pressure would correspond to the pressure force necessary for the production of the first palpitation of the pulse in the funnel-shaped

vein in the depth of the disc of the optic nerve, in the distant part of the vein where the pressure in that vein must be the lowest.

Has therefore the first palpitation of the " false " pulse that signification? A straightforward answer is: a pressure force able to exercise even a little pressure on the venous wall must exceed the pressure in that vein. This ideal equilibrium between the pressure in the vein and the impetus of pressure is not possible to be determined by the aid of any factor; it is therefore necessary to load the frame, as it were, by a trifle more weight to incline a little to one side in order to produce the first artificial movement of pulsation, that is to say, by giving way to the venous tube. For the purpose of accuracy of the result read on the Bailliart scale it would be necessary to add the pressure exerted by the increased tension of the eyeball due to cardiac systole, and also the forces causing the first throb of pulsation in the vein. These forces can be calculated from the rhythmic beats recorded by a Schiötz's tonometer, which amounts to, more or less, 2 mm. Hg to 3 mm. Hg. However, all instruments do not indicate the systolic throb in eyeball pressure. In this way, the above-mentioned investigation gives us results which are unquestionably a trifle too high as regards venous blood-pressure, but, in my opinion, the error is not very great.

When measuring venous blood-pressure in the eyeball where spontaneous " venous pulsation " exists, it will be necessary, first of all, to lower the pressure in the eyeball, which, as we shall find, is not a difficult matter. Measurements, however, on a soaked eyeball cannot, for obvious reasons, be accurate.

There is no doubt that the only measurements that possess a value are carried out on an eyeball having lower pressure than the venous blood-pressure or, expressed otherwise, where the venous blood-pressure is much higher than the pressure in the eyeball. Above all, the application of Bailliart's method can give positive results in cases of inflammation of the ocular nerve and of choked disc (papilloedema), due to increased central pressure.

That the venous blood-pressure is in a fixed relation to the central pressure, and that it is possible in each case to calculate the central pressure from the venous blood-pressure, as proved by Baurmann and Sobanski, about which, however, I find it difficult to express my opinion, and I will therefore abstain from dealing with these problems. Such rigid relations between the eye and the brain, that is to say, between venous blood-pressure and pressure on the cerebro-spinal canal would, I think, be rather unlikely to meet in a living organism.

Summing up the above results, it would be necessary to prove as follows: The " false " venous pulsation in the retina is always

passive, apart from what is due to interior pressure and cases when the pressure is " spontaneous." The extreme cause is always a rhythmic palpitation due to central eyeball pressure. The blood wave in the vein is uniform, the blood stream even and without palpitation due to pressure, and it is not therefore possible to ascertain the difference between systolic and diastolic pressure. Instead of this, it is possible to measure by Bailliart's method, more or less, the existing value in the interior vein, in so far as the pressure exceeds that in the interior of the eye, that is to say, in so far as there is no independent " false " pulsation.

I will now pass on to describing arterial pressure. As I have already mentioned in the foregoing, it is necessary to distinguish physiological pulsation depending on arterial pressure movements caused by " pulsation " as a result of pressure. The physiological pulse in the eye is a manifestation well known since long ago, but interpreted quite erroneously, and is, indeed, due to diseased changes in the arterial system, *e.g.*, in aneurysm of the aorta, in insufficiency of the valves of the aorta, and in Graves' disease. But even in these pathological cases there has been nothing further added to the knowledge of this phenomenon. Indeed, this phenomenon has become the order of the day. As stated in the foregoing, it is suggested by the theory on the pulse that the artery must dilate and contract. Should we be unable to perceive this, it is *not* the pulse. The arterial pressure movements, the *true* pulse, are a manifestation unknown as a factor. As a direct consequence of this, there occurs daily a succession of arguments among ophthalmologists hypnotized by the theory on the pulse. As I have indicated before in Part I of my article, we perceive these arterial pressure movements, by selecting a suitable method of observation, in, more or less, 80 per cent. of ocular examinations. By no means, therefore, does the circulatory system in pathological cases serve our purpose in this respect.

As I have already emphasized, we find that also in cases of a rapid pulse in insufficiency of the valves of the aorta, there result these pressure movements in a slightly different way, which is an exact proof that they express the physiological pulse.

Only the physiological pulse, therefore, which is entirely analogous with the pulse of the arterial system, can be an indication of retinal arterial pressure. For the carrying out of measurements, however, it will be necessary, as already stated, to exercise pressure on the eyeball artery in order to observe when the arterial pulse diminishes (diastolic pressure) and when it ceases (systolic pressure). This for easily comprehensible reasons is impracticable, not only because of technical difficulties in accomplishment, but also owing to the difficulty in perceiving this pulse.

I am not aware of the behaviour of the physiological pulse in the animal eye, as I have not carried out any investigations to that effect.

In consideration that pressure exercised on the eyeball artery is impracticable, it would perhaps be apt to question whether there is any pressure on the *eyeball* to replace it. Under such pressure it would be possible to observe the physiological pulse with accuracy, both its weakening and its disappearance. This method would not give the anticipated results. By exercising pressure on the eyeball, we change the physiological conditions of pressure ruling in the system of retinal blood vessels. The *physiological* pulse cannot therefore, so far as it can be judged, in connection with pressure on the eyeball, be an indication of physiological pressure in the retinal artery.

There still only remains the " pulse " due to arterial pressure. Can *this* serve the purpose of a factor for ascertaining arterial pressure, and therefore be the pulse suggested as such by A. v. Graefe nearly a century ago, and even to-day passes as such? In other words, does the result due to pressure on the arterial " pulse " permit us of ascertaining in the retinal artery physiological systolic and diastolic pressure?

But, above all, it is necessary to ascertain what this pulse is and how it behaves.

As we have already repeatedly stated, the pressure " pulse " does not in any way remind us of the physiological pulse. It consists of two phases: The first phase, arterial contraction, and the second phase, arterial dilatation. The first phase is rapid, the second slow. A. v. Graefe gives a definition of arterial systole and diastole which coincides with the definition given of cardiac systole and diastole. No doubt, the arterial movements remind us of the functions of the heart. But whence this sudden resemblance of the artery to the heart, and this at one particular spot in the vicinity of the disc? The contraction and dilatation of the blood vessel depend on the sympathetic nerve. Exactly the same phenomenon can be observed in the ears of rabbits. Due to excitation of the nerve, the artery contracts, and when this excitation ceases—it dilates. However, the rhythmic movement of contraction and dilatation due to cardiac systole and diastole, and also in pressure " pulse," we do not observe anywhere under the influence of *nerves*.

Is therefore arterial pulse due to pressure? As in cardiac systole, we see that there appears a gliding movement in the artery, which, subsequently, during the diastole, returns to its original position. We notice the same thing in subcutaneous arteries as we stated in the first place.

It would follow from v. Graefe's interpretation that from the moment when the cardiac systole takes place, the blood stream reaches the retinal vessels by overcoming obstacles in its way. This would mean that the blood stream must induce arterial dilatation, which, later, gives way to contraction during the cardiac diastole.

In the meantime we have proved that the artery gives way to contraction during cardiac *systole*, and *dilates* during the diastole, the former phase, *i.e.*, the arterial contraction being rapid, and the latter phase, *i.e.*, the arterial dilatation, slower. It cannot so far be ascertained whether the wave making its appearance due to cardiac systole is the original cause of the throb of the arterial pulse, because this throb depends on the *contraction* of the artery. If the systolic wave from the heart were to be reproduced, the first condition would be a *dilatation* of the artery.

The arterial " pulsation " evident at pressure cannot be anything else than passive pulsation, and is therefore a " false " pulsation produced in the same way as " false " venous pulsation with rhythmic pressure palpitation in the eyeball.

In evidence of my proof, I quote an apt remark with which I have not hitherto met with references in the literature.

It refers to congestion of the central retinal artery. In a case of this kind where there occurs pressure on the eyeball, we observe, at the same time, arterial " pulsation." This " pulsation " is altogether identical with pressure pulsation in a normal eye. However, it embraces only a small part of the entire arterial region. *In an artery, therefore, not reached by the blood stream, because it is plugged off (to a great extent), there can be produced " pulsation."* This " pulsation " can only be passive " pulsation " as resulting from pressure palpitation in the eyeball.

Such also would be the state of things in connection with the problem of the so-called arterial pulse. We are now faced with the following problem: This manifestation of arterial pulse somehow cannot indicate to us the amount of diastolic and systolic pressure, because—as I have pointed out—this difference is immeasurable, and, in any case, must be an expression of the pressure conditions in the arterial system of the eyeball. The result obtained by Bailliart's apparatus shows us the impetus needed to exert pressure on the central retinal artery. Can this measured force also serve as a measurement of pressure in that artery?

Wishing to give a reply to this question, let us first examine the mechanism of the actual state of the pulsation, not by beginning with the final phase, but from the very commencement of the experiment undertaken. By exerting pressure on the eyeball, we first observe the " venous " pulsation in the vascular funnel, in so

far as there has not already occurred pulsation previously. When measuring the pressure, we close the vein.*

At the same time, the disc becomes colourless (this is not quite visible). The artery begins to " pulsate," and in a number of instances these " pulsations " have also included the vein, usually in alternation with the artery.

In the first place, therefore, the venous tube is closed. Although this conclusion is unquestionably imperfect, it suffices to check the normal outflow of blood, the vein in question being a terminal vein. In consideration of the fact that the outflow has been checked, it is necessary to discontinue the incoming flow as well. This, however, need not be proceeded with immediately, due to the capacity of the venous and precapillary-capillary system. The latter, however, is also subjected to pressure. A corresponding pressure of very much longer duration is required by the retinal artery to reach the pressure in the ophthalmic artery. However, any blood stream must not make its appearance in the central artery. Between the tightened vein and the central eyeball arterial branch of the ophthalmic artery, the blood stream will either stop completely or move on very slowly, forcing itself through the tightened vein with great difficulty.

By now increasing the pressure force on the eyeball, there begins, at a certain moment, to reveal itself the first throb of pulsation in the artery. The force to be used in order to produce these manifestations, *i.e.*, the pressure measured on the basis of

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* The closing of the vein by pressure on the vascular funnel is comprehensible, as it is performed on the spot where the vein possesses the lowest pressure. On the other hand, it is more difficult for us to understand why *arterial* pressure becomes evident in the vicinity of the vascular funnel. The pressure must first be exercised where there exists the lowest pressure, namely, in the ramifications of the arterial circulation.

According to the laws of hydrostatics, the pressure exercised in the closed vessel spreads uniformly to all vascular walls. This pressure should therefore reveal itself first in the ramifications of the arterial circulation wherever the resistance happens to be the lowest. Why such is not the case is hard to understand. Why is the region in the vicinity of the disc more yielding to pressure, and why is pressure not exercised uniformly on all arterial ramifications in that region? Here, indeed, we are confronted with questions which only can be answered conjecturially. One might, of course, be able to prove that the minor arteries possess more power of resistance than the major ones. It should further be indicated that we also measure the reaction to blood pressure by the resistance of the vascular walls the value of the resulting measurments of which in regard to blood pressure, notwithstanding the inclusion of factors of resistance as suggested by Fritze, has been very much minimized.

It is difficult to be satisfied with the universally accepted view on pressure by forcing a glassy substance into the vascular funnel in order to produce " pulsation " and to check the veins. By that pressure we measure uniformly the outflow on every square millimetre of the wall of the eyeball. The pressure, therefore, on the vascular funnel exceeds that on any part of the retina. However, the problems which I have just indicated, are not very essential. Yet, after all, it is a correct deduction, and, at any rate, it is shown that pressure is capable of giving rise to " pulsation " and to contraction of the blood vessels.

Bailliart's scale plus the eyeball pulsation, indicates to us the pressure in the ophthalmic artery, but not the pressure exercised in the central retinal artery. The former we measure according to Bailliart's scale, and the latter can be calculated, as I wish to mention here, by the rhythmically moving pointers on Schiötz's tonometer (2—3 Hg).

It would still be necessary to explain the simultaneity and alternation of arterial and venous " pulsation," which sometimes occurs due to contraction of the central vein. I believe the matter is simple enough provided that the arterial pressure and checked vein are in harmony, and if so, the pulse in both vessels is simultaneous, provided, of course, that the pressure differs in both or, in other words, that there is an alternating " pulse," *i.e.*, the same movement in the intra-ocular pulse, first, vascular contraction at a minimum of tension, followed by contraction at increased tension.

Moreover, the " pulse " manifestations and the relation between the arterial and the venous " pulse " may depend on different factors, such as, for instance, the anatomical vascular system, the pressure rate, etc. Should the pressure in a given vein be very high, then any " pulsation " in that vein is entirely out of the question. However, wherever a " pulse " occurs, it is entirely passive, that is to say, produced by an exterior force acting on the vessels. By summing up the above results, I am able to state the following:—

When exercising pressure on the eyeball, there occurs a contraction of the central vein, and due to this circumstance the flow along the whole extent of the central artery as coming from the ophthalmic artery through the medium of capillary vessels, encounters complete restraint or, at least, a substantial check. The blood stream arrives at a standstill. There now results an equilibrium between the pressure in the ophthalmic artery and that in the central retinal artery. In proportion to further pressure, there occurs in the artery a " pulse " consisting of two phases. The first rapid phase is due to arterial contraction, and the second more lasting phase to arterial dilatation. These movements are passive, being produced by the throbbing pressure in the eyeball. This is exactly the same thing as happens in venous " pulsation." The force needed in order to produce the first throbbing " pulse " consists of three factors: (1) eyeball pressure; (2) pressure exercised by Bailliart's apparatus, and (3) pulsation of the eyeball artery. As regards Bailliart's method, we can therefore measure with more or less accuracy the pressure in the ophthalmic artery, but not in the central retinal artery.* The difference between

* Seidel has also written on this point.

the pressure in the ophthalmic artery and that in the central retinal artery—we must confess—is known near enough, seeing that the central artery is considerably narrower than the ophthalmic artery, and, according to the laws of physical pressure applied to the liquid passing from a wider tube to a narrower one, it diminishes considerably. Thus the dynamometer test affords us the possibility of determining the approximate pressure in the ophthalmic artery, but in any gradual pressure it differs from that in the central retinal artery for the determination of which we have no means at our disposal.

Summing up all our results in brief, we are in the position to state as follows: The arterial "pulse" as well as the venous "pulse" is a passive pulse produced by a rhythmic throb due to eyeball pressure. The blood stream in the vein is uniform, circulating without any oscillations due to pressure, so that there is no difference between diastolic and systolic pressure. Due to a fundamental error of Bailliart's method, pressure pulse has been identified with spontaneous pulse. A spontaneous pulse in the retinal artery comes nearer the truth. It can be observed in 80 per cent. of quite healthy individuals who are not suffering from circulatory disturbances. The result turns out to be different in cases of rapid pulse. This depends on the rhythmic onward thrust of a section of the artery, due to cardiac systole, in the same way as the pulse in other arteries. However, only adequate experience in the observation of spontaneous pulse could afford us an answer to the question of pressure in the retinal vascular system. Unfortunately, such experience is humanly impossible.

The methods of investigating pressure in the brachial artery cannot be adopted for the measurement of pressure in the retinal blood vessels. The pressure on the eyeball creates entirely different circulatory conditions, so that there is no analogy between these two methods. Considering that the physiological arterial pulse is hardly perceptible, the difference between systolic and diastolic pressure is practically nil, and is therefore not capable of being measured. If conditions were such as suggested by Bailliart and others, *i.e.*, 0·45 : 1, the spontaneous pulse in the retinal artery would necessarily be seen in every eye at once, and would necessarily be more distinct than the above mentioned arterial pulse, where this proportion amounts to 0·6 : 1.

By Bailliart's method we are able to investigate the height of retinal venous pressure, in so far as the vein does not present independent "pulsation," *i.e.*, "pulsation" occurring already without pressure. Bailliart's method does not enable us to estimate pressure in the central retinal artery, but we can thereby

determine the approximate pressure existing in the ophthalmic artery.

In consideration that the central retinal artery is considerably narrower than the ophthalmic artery, the pressure in it must necessarily be considerably lower, which, however, cannot be determined even approximately.

PTERYGIUM*

BY

EMANUEL ROSEN

NEWARK, N.J.

THE great number of operations devised for pterygium, like the many operations proffered for ptosis, bespeaks the frequency of failure in this surgical condition. Recurrence in pterygium operation is the rule in the hands of the experienced, as well as in those of the novice. Having seen a great many pterygia in the past 2½ years, among which were included a rather large number of recurrences, it became increasingly apparent that the usual method of surgical treatment, namely, the McReynolds transplantation, was not functioning too well in the hands of many ophthalmologists. This procedure was the operation of choice of many ophthalmologists with whom I spoke and most men readily agreed that recurrences were all too common. Each seemed to have one modification or another, but all used the McReynolds operation as the basic procedure. It is true that in a good many cases a follow-up was rather difficult, so that the final result was not adequately obtained. McReynolds[1] has stated that his operation was a modification of the Desmarres' procedure, the feature which he introduced being the closure of the exposed sclera following the separation of the pterygium. McReynolds believed that if a break in the conjunctiva occurrred in the axis of the palpebral fissure, the results would be more or less unsatisfactory, for the insertion of sutures bringing the divided parts into apposition is bound to produce some thickening, and irritation consequent upon these conditions will serve to excite the neighbouring subconjunctival vessels, and thus cause a return in growth of the pterygium. " By concentrating the vascular activity underneath the lower lid where the pterygium is not only removed from view, but protected from irritating influences of dust and exposure, the process of atrophy naturally and

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FIG. 1.

Shows a typical moderately advanced pterygium of characteristic appearance before operation.

FIG. 2.

Shows the same eye approximately two months after operation.

surely follows." It does not seem justifiable to assume that conjunctival approximation cannot be attained adequately in the midline, for in a great many operations the site of union is soon not recognizable. Again "vascular activity need not be concentrated in the lower cul-de-sac"—burial into the squamous-like caruncle would seem to be a much more compatible histological union. McReynolds also emphasized the importance of complete removal of the head of the pterygium with a sharp knife, suggesting that any technique of divulsion should be avoided. The knife should be as sharp as possible and no attempt should be made at tearing the pterygium head from the cornea. This point is worthy of re-emphasis, especially in view of a recently reported operation in the Navy Medical Bulletin[2].

Pterygium operation

Technique.—The head of the pterygium is grasped with forceps as in any operation for pterygium, and is carefully dissected off the cornea to the limbal area with a sharp knife. The conjunctiva is then separated from the limbus both above and below the pterygium for a distance of three mm. and undermined from the limbus to the region of the newly retracted pterygium head. (It seems that upon undermining the conjunctiva in this area after separation at the limbus, the pterygium also retracts). A double-armed suture is then inserted about 1 mm. from the head of the pterygium going from conjunctival to episcleral surfaces with the thread coming into position vertically. The pterygium is then folded back upon itself so that episcleral tissue is in contact with episcleral tissue and the double-armed suture is brought out through the centre of the caruncle, the needles coming through at 2 mm. vertical separation. The caruncle is punctured with some difficulty, this procedure being slightly painful. The episcleral tissue below the pterygium should be undermined carefully and completely, so that no adhesions hold back the folded pterygium. The suture is then pulled tightly and tied over a small rubber button. This process of folding the pterygium back upon itself produces a tendency toward knuckling of tissues which varies with the broadness of the central portion of the pterygium, and is the one objectionable feature. However, if the pterygium is quite broad, after the head is dissected off the cornea two parallel incisions may be made in the pterygium base separated by a distance equal to the width of the mid-head of the pterygium, and running horizontally for several mms. before folding the pterygium back upon itself. The edges of the conjunctiva are sutured together in a horizontal line starting from the buckled end of the pterygium and going to the limbus. The last conjunctival limbal suture should be placed very carefully and cut quite closely so that no suture ends impinge upon the cornea. 6-0 silk should be used for these sutures. Usually three or four sutures are required to close the conjunctiva completely.

This procedure is adequate and not at all difficult to perform in the mild types of pterygium, but may also be used in the more disturbing recurrent variety. The procedure is much the same although in one instance it was found to be of distinct advantage to use two double-armed sutures, one being brought out at the caruncle and a second coming through the structure of the pterygium itself. The small rubber button prevents the suture from sliding back into the folds of the conjunctiva where difficulty

FIG. 3.

This is a diagrammatic representation of the various steps in the operation for pterygium. (*a*) Characteristic pterygium before operation. (*b*) Pterygium being severed from the cornea and dissected back to its base. (*c*) Double armed suture placed through pterygium head and emerging from the episcleral side. A small area of conjunctiva is undermined at each limbal area and appears like a small crescent. (*d*) Each arm of the suture is passed underneath the pterygium and brought out through the caruncle. The pterygium is separated from the sclera with the scissors at its upper and lower borders. (*e*) The double armed suture is tied over a small rubber button. The upper and lower free edges of the conjunctiva are sutured with three or four single sutures. (*f*) The conjunctiva is completely approximated.

is usually encountered in removal, and frequently requires cutting down into the conjunctiva. This suture may be retained for ten days. The other conjunctival sutures usually work out readily. A moderate amount of secretion exists for several days but may be handled adequately through the use of hot compresses. The patient experiences some discomfort for two or three days and the operation should be regarded as a major procedure for that post-operative period.

During the past seven months twenty-five cases have been treated in this manner, in which time there has been only one recurrence. These cases have only been followed from two to six months, but in many instances the appearance at the end of two months seems to indicate that there will be no recurrence for the caruncle flattens out, the conjunctiva unites evenly and the opaque cornea becomes thinned out. In the single instance where the result was not considered very satisfactory, the patient had previously been operated upon twice and the involved tissue was hypertrophied and fleshy. There was overlapping and redundancy of tissue after the pterygium was folded back upon itself. Two weeks after the operation the redundant tissue was cauterized with silver nitrate and several days later the appearance was regarded as most satisfactory.

The advantages offered by this type of operation are as follows:

1. The head of the pterygium is brought in contact with the modified cutaneous structure, the caruncle, a structure with which the stratified pterygium appears to be compatible.

2. Episcleral tissue tends to fuse with episcleral tissue (produced by folding of pterygium).

3. The direction of growth of the pterygium is completely reversed.

4. An interposing bridge of conjunctiva is placed between the folded pterygium and the corneal focus.

5. Undermining the conjunctiva at the limbus tends to relax the pull upon the pterygium and prevents overhanging of the conjunctiva at the limbus.

6. Since temporal pterygia are extremely rare they need not be considered.

7. The pterygium is completely buried and is no longer exposed to direct elemental irritation.

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ANTERIOR LENTIGLOBUS
An Atypical Case *

BY

K. SEN

CALCUTTA

ANTERIOR lenticonus is a very rare condition. It consists of a small conical projection of the lens at its anterior pole.

Anterior lentiglobus is also very rare. It consists of a small globular projection of the lens at its anterior pole. The projection is a part of a sphere and in lenticonus it is part of a cone.

In both cases the projection consists of clear cortex only. The nucleus remains intact and undistorted (Feigenbaum, 1929; Kienecker, 1929). The projection takes place after birth otherwise the nucleus of the lens would have been affected; however, Seefelder and Wolfrum (1907) noted it in a foetus of 4 months. The projection is in the pupillary area. It usually occurs as an anomaly without any other changes in the eye.

The lens itself is transparent. On examination with the ophthalmoscope a dark disc is seen in the centre of the pupillary area

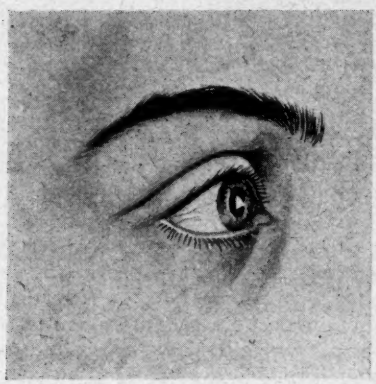

FIG. 1.

Anterior lenticonus (Duke-Elder).

" resembling in appearance the effect produced by an oil globule in water: this is due to the fact that none of the rays from the fundus reaches the observer's eye owing to prismatic reflection in the axial region " (Duke-Elder, 1938), Figs. 1 and 2. As the projection has

* Received for publication, September 23, 1947.

FIG. 2.

Anterior lenticonus as seen by the plane mirror of the ophthalmoscope (Duke-Elder).

an increased curvature the area is highly myopic (−20·0 D., Kienecker, 1929). The projection has always a tendency to increase. Feigenbaum (1932) followed up a case for six years and recorded that the projection increased in curvature making the area more myopic, the increase being from −5:50 D. to −10·0 D. As long as the capsule remains intact the projection of the lens remains transparent. Eventually an anterior polar cataract develops (Jaworski, 1910; Tsukahara, 1930; Feigenbaum, 1932), most probably due to the rupture of the lens' capsule from overstretching.

The cause of this condition is not definitely known. A delayed separation of the lens' vesicle (Krusius, 1910), an inflammatory adhesion to the cornea (Mohr, 1910), have been suggested. Collins (1910) suggested that the normal suspensory ligament in its development exerted chief traction on the anterior capsule and the absence of such traction might account for anterior lenticonus. Mann (1937) noted that in some lower animals the lens appeared to bulge through the pupil as though the iris was pressing it back at the periphery, and suggested that it was possible that some such deforming stress might have occurred in foetal life owing to a too rigid pupil so that the lens had been permanently moulded.

The following case shows some very unusual features.

A. S., a Sikh boy, aged 14 years, was seen on October 1, 1946. He complained that on waking up on the morning of September 28, 1946, he discovered that he did not see well with his right eye. He was a tall, well developed boy. His physical examination revealed no abnormality. His father was seen and he showed no abnormality.

On enquiry from his father it was noted that there was no abnormality in the bodies and eyes of the boy's mother, brother and two sisters. There was no history of trauma to the eye of the boy.

On examination of the right eye the conjunctiva, cornea, and anterior chamber were normal. Pupil was reacting well to light and accommodation. There was a very small opacity on the anterior surface of lens at its upper and inner quadrant and the opacity was mostly covered by the edge of the pupil. The central part of the pupillary area seemed to be quite transparent. The tension was normal. The vision in the right eye was 5/60 and

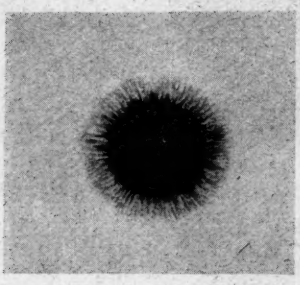

FIG. 3.

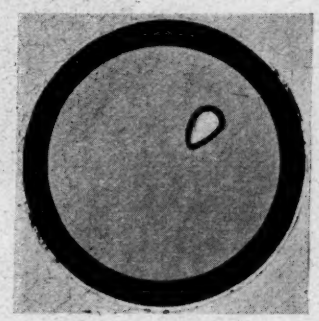

FIG. 4.

there was no improvement with glasses, but on contracting the pupil with eserine the vision improved to 6/24 (+1) with −2·0 D. The vision in the left eye was 6/6 with −2·75 D.

On dilating the pupil the size of the opacity was found to be approximately 3 mm. by 2 mm. (Fig. 3). On examination with lens and loupe the small opacity on the lens was found to be really a tiny little projection of the anterior surface of the lens; the centre seemed to be transparent and the margins were grey. It was egg-like in shape, its longest axis being in the 1 o'clock—7 o'clock meridian, the broad part being towards 1 o'clock and the narrow end towards the centre of the pupil. With transmitted light (Fig. 4) the centre was found to be transparent and the edges were opaque. With slit-lamp (Figs. 5 and 6) it was found that the projecting part consisted of a herniation of the anterior cortex of the lens through an egg-shaped rupture of the capsule the edges of which could be seen surrounding the herniation and were curling forward. The capsule over the projecting part was thought to be deficient. The surface of the projection was irregular and was pitted at the lower and outer part. There were several lines of tension on the capsule of the lens from the narrow end of the ruptured capsule spreading

over the pupillary area. This probably explained why the vision could not be improved beyond 6/24 (+1). The curvature of the projection was spherical and not conical. The other parts of the lens including the nucleus were normal There were no myopic

FIG. 5.

FIG. 6.

changes in the fundus. There were no other abnormalities in the eye either congenital or acquired, inflammatory or abiotrophic.

A short description of the case and the paintings of the lens condition were sent to Prof. Ida Mann, and she has very kindly sent the following remarks, " Embryologically I cannot explain it at all, though I do feel that one cannot be certain of the complete absence of the capsule over the bulge. If this had been the case I should have expected the lens fibres to have become opaque and partially dissolved. I think therefore that there must be a very thin layer of the deep lamella of the hyaline capsule and it is the zonular

lamella which is absent over the bulge and everted around the base. What could have produced this I have no idea, though probably trauma at or soon after birth might account for it."

The case was again seen in January, 1947, *i.e.*, three months later and the condition was found to be exactly the same. No opacity of the lens cortex had developed and there was no increase in the size of the projection. The vision was still the same.

Discussion

Embryologically the lens capsule is composed of two layers. The deep layer is secreted by the cells of the lens vesicle and the superficial or zonular layer is added later on the outside of this in the

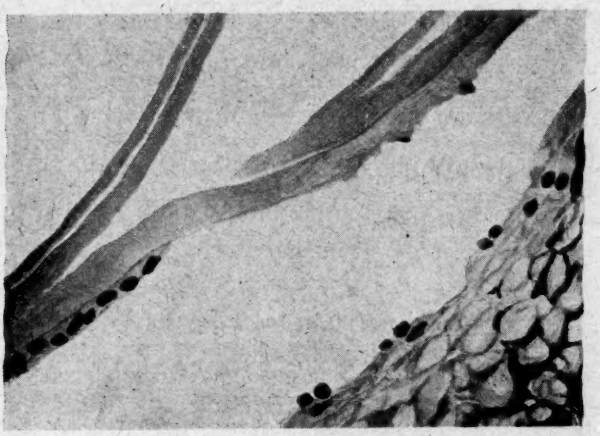

FIG. 7.

The lens capsule (Tooke)

peripheral part and is formed by the coalescence on the lens of the fibres of the suspensory ligament (Mann, 1937). Histologically two layers can be demonstrated either by staining with aniline blue (Beauvieux, 1922) or by use of a silver impregnation method (Busacca, 1929). Tooke (1933) demonstrated that each of the two parts of the capsule was divided into separate layers (Fig. 7). Vogt (1925–32) and Elschnig (1929) believed that the zonular lamella extended completely over the anterior surface, while Busacca (1929) claimed that there were three different structures, a zonular lamella confined to the lateral portions of the lens, a fine pericapsular membrane surrounding the entire lens and a capsule proper composed of several layers.

In this particular case, the lens capsule, except a very thin lamella of the deep layer, was ruptured and the edges were curling forward. Although under the slit-lamp no capsule could be demonstrated over the herniated area (Fig. 6), a thin layer must be present otherwise, as Prof. Mann pointed out, there would have been opacification and partial absorption of the lens fibres.

The curvature of the projection was regular and spherical. The surface of the projection presented a most interesting appearance (Fig. 5). The surface was pitted. This was specially apparent at the lower and outer part of the projection. This appearance has not been seen or described in cases of exfoliation of the zonular layer of the lens capsule. This appearance is very difficult to explain.

Too much reliance cannot be placed on the history of sudden onset of dim vision as many cases even of amblyopia ex anopsia state that the dimness of vision in the particular eye is of a few days' or weeks' duration.

In the absence of any other abnormality either congenital, acquired or abiotrophic, one would like to suggest that the condition was due to a congenital weakness of the lens capsule and most of the layers gave way either spontaneously or during a very trivial trauma allowing a herniation of the anterior cortex. It does not seem possible to postulate when the rupture took place.

Summary

A case is described in which there was a tiny herniation of the anterior cortex of the lens through an egg shaped rupture of the superficial layers of the anterior lens capsule.

The cases of anterior lenticonus and anterior lentiglobus described in the literature showed the projection of the anterior cortex to be either conical or globular in shape. They were round and situated at the anterior pole of the lens occupying the pupillary area. The capsule over the projection was intact especially at the early stages. In this case, most of the layers of the anterior capsule of the lens were already ruptured and the edges were curled forward. The herniation of the anterior cortex was egg-like in shape, was eccentric in position and the surface was pitted.

It seems that a case like this has not previously been reported. It was intended to report this case after keeping the patient under observation for at least one year. When he was sent for in March, 1947, it was found that he, with his family, had left for his home in the Punjab. In July, 1947, he or his family had not returned and their whereabouts were not known. (Note.—Severe communal riots broke out in the Punjab in March, 1947).

All French and German references have been taken from Duke-Elder's Text-Book of Ophthalmology.

Finally, thanks are due to Major E. J. Somerset, I.M.S., for help in preparing the paper and also for some useful suggestions.

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ON GENESIS AND OPERATION OF SENILE ENTROPION

BY

Prof. A. KETTESY

DEBRECEN

CONTRARY to cicatricial entropion with anatomical changes of the intermarginal surface and of the tarsus, senile (or, as it is sometimes called, spastic) entropion is a simple turning in of the unchanged tarsus of the lower lid.

Under normal conditions the lower lid (correctly its skeleton: the tarsus) is kept in right position by two forces: (1) by the elasticity of the tarso-orbital fascia with the embedded inferior tarsal muscle; (2) by the tone of the orbicularis muscle exerting equally distributed pressure upon the lid. The orbicularis muscle plays the more important rôle. Its normal distribution is maintained by connective tissue branching off between the bundles. Other forces such as capillary adhesion and elasticity of the skin are of less importance.

In senility there is sometimes a slackening of the whole palpebral connective tissue, creating a situation ready to result in entropion: drawing up and accumulation of the bundles of the orbicularis in the lid-margin. The lid is in this phase still in its normal position but unbalanced. Every small pressure at the lid-margin in simple

blinking, is apt to upset the equilibrium, when the tarsus is on a sudden turned in, taking with it skin, muscle, and eye-lashes.

We are able to reset the lid into its original position by simply pulling the skin downwards, thus bringing about normal distribution of the orbicularis muscle.

The inhibiting rôle of the tone of the inferior tarsal muscle was shown by Blaskovics. Instilling cocaine and adrenalin, we increase the power of the tarso-orbital fascia, consequently entropion cannot ensue. The result, of course, lasts only as long as the drug is working.

The majority of the operations for senile entropion act on a similar principle: they increase one or several of the various forces, that are keeping the lid in right position.

Surgical operations have been directed towards (1) shortening of the skin (Celsus, Terrien, Snellen, Hotz, Imre); (2) re-establishing the absent pressure on the convex tarsal margin (Graefe, Koster, Birch-Hirschfeld, Montgomery, Goldzieher, Blaskovics and others); (3) The same pressure is obtained by a triangular excision of the tarsus (Muller); (4) Increasing the tone of the tarso-orbital fascia by operative procedure (Tóth, Trantas); and (5) we might add the proceedings, that are combining several of the above mentioned (Blaskovics).

It is a common feature of all groups that they leave unattacked the decisive factor of the turning in: the accumulation of the orbicularis in the margin, or they touch it only secondarily and insufficiently, hence the relapse occurs mostly very soon, or, trying to get a lasting and satisfactory result by increasing the effect, the entropion turns into ectropion.

Out of the described mechanism ensues the fact that the accumulation of the orbicular muscle is terminal. Therefore, from the practical point of view this is the only cause. All the other conditions are only preliminary.

The solution of the problem is excision of the orbicularis muscle of the lower lid without anything further.

It was Hotz who proposed the removal of the palpebral part of the muscle first, as a detail of his well-known operation originally devised for cicatricial entropion. He excised only some bundles in order to make the tarsal surface bare for putting in his everting sutures.

Our procedure consists of a thorough excision of the muscle, the whole palpebral portion as well as the greater part of the orbital section.

Local anaesthesia. We put a lid-plate into the lower fornix. Pressing it against the lid, we are able to perform the operation in total bloodlessness.

The skin incision is close below the line of the eyelashes. We

undermine the skin downwards as far as the orbital margin, advancing between skin and muscle. The palpebral part of the orbicularis as well as the lower portion of the muscle is thoroughly extirpated with fine forceps and scissors until tarsus and orbital fascia are clearly exposed.

Sutures are superfluous, for coaptation of the wound is faultless.

This little operation gives immediate and lasting result. The muscle being absent, no relapse is possible. There are two parts of the muscle that remain intact: the limbalis and the Riolani, sufficient to maintain normal position and function of the lid.

Over-correction, a delicate point of lid-operations, is not possible. It is well-known that operations for entropion if at all efficacious, easily produce an ectropion, chiefly if there was excision of skin. Removal of skin is in our operation never necessary and therefore absolutely forbidden. Sometimes at the end of the operation the skin appears to be redundant, as it applies itself to the line of the eyelashes in folds. We are not persuaded by this sign to excise the skin, as the consecutive fine cicatrisation smoothes out the skin in a few days.

A NOTE ON THE PHYSIOLOGY OF THE AQUEOUS HUMOUR*

BY

E. BÁRÁNY *and* H. DAVSON

IN recent papers it has been shown (Bárány, 1947, *a*, *b*, *c*), that the rate of penetration of sodium into the aqueous humour is dependent to only a small extent on the blood pressure. The equilibrium concentration of sodium in the aqueous humour and the osmotic pressure of the aqueous are even less affected by a blood pressure reduction. These results have been discussed in relation to certain possibilities of the mode of entrance of sodium, namely :—

(*a*) Secretion in accordance with an equation based on certain assumptions (Kinsey and Grant, 1942).

(*b*) An ultra-filtration process involving an actual bulk movement of fluid.

The possibility of a simple diffusion mechanism as envisaged by Davson and Quilliam (1940), appeared to Bárány to be excluded by Kinsey and Grant's work. The conclusion was reached that bulk movement of fluid was a negligible factor in the penetration of sodium and in the formation of the aqueous humour generally and that the rate of flow of aqueous was only little affected by the intraocular pressure.

* Received for publication, September 24, 1947.

We should like to emphasize the dependence of these conclusions upon the validity of the interpretation by Kinsey and Grant of their own results, an interpretation not accepted by Duke-Elder and Davson. The papers by Bárány have been criticised recently by Davson (1947) in this light but there are a few corrections that must in fairness be made. The statement by Davson "The notion that a molecule or ion may not enter by simple diffusion but may enter by ultra-filtration has no physical meaning" is indeed ultimately true of any membrane system; nevertheless the conditions may be such that quantitatively the penetration of a substance by simple diffusion may be small in comparison with that entering by ultra-filtration. Thus, if salts could only penetrate the eye membranes in a strictly limited region and if, moreover, filtration from the blood took place only in this region, and consisted in a continuous flow into the eye, it could be stated that sodium entered predominantly or perhaps exclusively by a filtration process, simple diffusion being excluded by the impermeability of the major part of the membrane to this substance. Such a system was, in fact, envisaged by Bárány although this was not explicitly stated. Bárány's experiments actually exclude this mechanism since the rate of penetration of sodium was almost independent of pressure.

One other point should be mentioned. Davson objected to the use of a mean value not significantly different from unity. It is agreed that this value was not statistically different from unity but it should have been pointed out that exactly the smallness of this difference was in favour of the general argument.

In conclusion, perhaps it is worth devoting a few words to the general problem of whether salts are actually secreted into the eye or whether they may enter by simple diffusion. Kinsey and Grant's "secretion equation" is very similar in form to the simple diffusion equation of Davson and Quilliam; it is not impossible that their data would fit a correct dialysis equation equally well as they actually fit their "secretion equation." The "dialysis equation" of Kinsey and Grant must be unrealistic, as pointed out by Duke-Elder and Davson (1943)† and therefore it is not surprising that it does not fit their data.

The problem of the secretion of salts into the eye is thus still in a state of flux; it would appear that a mere mathematical analysis of the data already to hand is not sufficient for a decision.

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† According to a personal communication to Davson, Friedenwald is of the same opinion.

ANNOTATIONS

Bad Debts

No one can go through his professional life without accumulating a certain proportion of bad debts. Our personal feeling was one of surprise that these were not greater than they were, and it speaks well for the honesty of patients in general that with most of us bad debts are few. Patients of the type of Rawdon Crawley, the arch-exponent of how to live on nothing a year, are always bad debts. Some cases, however, are perfectly genuine instances of the defaulter not having the means to settle his account, owing to financial depression, some sudden calamity or other cause. It might be argued, and with justice, that he should not have chosen that particular moment to consult his oculist. Occasionally the unpaid account is due to sheer laziness and unbusinesslike habits on the part of the recipient. Such people can be brought to book by employing a debt collecting agency or sending a solicitor's letter. But the hard baked non-payer is usually proof against even these. He does not mean to pay and the only way to make him is to institute legal proceedings. For small sums, such as a single consultation, it is obviously not worth while to go to law. Even if the amount is larger, it is doubtful whether the publicity may not do more harm than good in the long run. Our own view was that these people were not worth powder and shot. In one instance we obtained payment of a long standing account by refusing to see any other member of that family till it was settled. The loss of this type of patient does not hurt one's practice.

The patient who pleads poverty and manages to get his surgeon to accept less than the usual fee for an operation is not quite in the same class as a bad debt. We recall an amusing example of such a case in our early days when we were asked to assist at an operation for cataract. On us devolved the duty of instilling cocaine into the eye of a little rat-like personage, who sat on a chair in the corner, in a state of profound melancholic abstraction. Once or twice he looked up at us with his other eye in which we observed a peculiar twinkle of satisfaction. He was a cheap case and his surgeon had learnt, after the question of fees had been settled, that he was really quite well off. This must have been the cause of his twinkle of satisfaction. "I've done you fellows in the eye and am going through with it" was what we imagined it conveyed.

Of other bad debts we remember a young man who had a small corneal abrasion following injury from a tennis ball. He was shocked out of all proportion to the extent of his injury, and we had to provide him with a whisky and soda as well as the appropriate local

treatment. We never saw him again and he never paid his fee and got away with more than one expects to do when visiting a professional man.

Amicum perdere est damnorum maximum

The Latin tag which forms our heading says that to lose a friend is the greatest of losses. Among a professional man's oldest friends are the tools of his daily work. The ophthalmoscope one has used for many years is a very old friend indeed and it behoves us to take care of it. We well remember the annoyance of a friend of ours at the loss of the ophthalmoscope he always used. It was a Frost's model and had seen much service. He was going home one afternoon after the out-patient work at hospital was over, and he travelled on the top of a bus. The ophthalmoscope was carried in the tail pocket of his morning coat. Some miscreant picked his pocket and he arrived home without his ophthalmoscope, It was a grievous blow, and it took him some time to get used to a substitute. The moral of this story is that you should carry your tools either in an attaché case or in some pocket not so easily picked as one in your coat tails, if anyone ever wears a morning coat nowadays. Mr. William Lang always carried his ophthalmoscope loose, in sections, in his waistcoat pocket and we never heard of his losing it. The other alternative is to eschew travelling on bus tops.

BOOK NOTICES

The British Orthoptic Journal. Published annually by Wilding and Son, Ltd., Shrewsbury. Price, 7/6.

The fourth volume of the *British Orthoptic Journal* maintains the high standard set by its predecessors.

It opens with an editorial note describing certain innovations in the *Journal*, such as the invitation to ophthalmic surgeons and to orthoptists overseas to contribute articles and it closes with lists of successful candidates in the 1947 examinations and of the various orthoptic training schools. The body of the *Journal* is made up of some twenty-eight separate papers written by ophthalmic surgeons and orthoptists, a copy of the general and of the detailed syllabus for orthoptic students and an account of the activities of the British Orthoptic Society and of its branches.

The papers are remarkable for their general excellence and for the variety of subjects with which they deal, e.g., "are orthoptics really

necessary"; "accommodative squint associated with emmetropia"; "the adoption of rhythmic illumination for orthoptic treatment"; "a new method of developing normal retinal correspondence"; and "some observations on operation results." There seems to be general agreement among orthoptists that the period of treatment should not be unduly prolonged before the aid of surgery is invoked and in one paper, by an ophthalmic surgeon. it is written that the orthoptist might suggest to the surgeon which muscle or muscles it would be advisable to operate upon. The author says that many surgeons would disagree with the idea but he justifies it on the grounds that the orthoptist has a more detailed knowledge of the case than has the surgeon.

Another point of general agreement seems to be that the period of clinic treatment should be shortened as far as possible. In some papers we are told how to separate the cases which are likely to benefit from orthoptic treatment from those which are not, others deal with the value of homework as supplemental to training received in the clinic and others with procedures for overcoming suppression and abnormal correspondence more quickly than is usual when employing standard methods.

In general, this volume provides evidence of the progressive nature of orthoptics and of the keeness and enthusiasm of its practitioners and we hope it will enjoy the large circulation it deserves among ophthalmic surgeons.

Chronic Ill-health relieved by drainage of the para-nasal sinuses. Rosa Ford. Pp. xii and 104. Henry Kimpton, London, 1948. Price 6/-.

A list of the diseases which the authoress states have been cured or relieved by suitable drainage of the nasal sinuses is, to say the least, formidable. In some ways it is reminiscent of the more adventurous and enthusiastic lucubrations of the advertisers of patent medicines, ranging as it does, from amenorrhoea to strabismus, and from obesity to disseminated sclerosis. Indeed the very universality of her claims does her some disservice, since the chapters on diagnosis, pathology and treatment are reached only after wading through a series of case reports, in which sinus infection, not diagnosed by orthodox clinical methods, is presented as the ultimate cause of diseases for which no other origin can be found, and its treatment as the ultimate panacea.

Reports of forty-seven cases are given in brief; the story is similar in all; ill-health of various kinds, fully investigated with no cause being found; sinusitis diagnosed on the strength, in most cases, of contraction of the visual fields; treatment by inhalations, 50 per cent. glycerine drops, and hydrogen peroxide nasal swabs; discharge from the nose; amelioration or cure. The book may

perhaps do some little service in drawing attention to the possibility of hidden sinus infection as a possible aetiological factor, but the authoress's criteria of its presence, and indications for treatment will hardly command universal approbation.

OBITUARY

R. D. BEATSON HIRD, M.D., F.R.C.S. (Ed.)

OPHTHALMOLOGY in the Midlands suffered a great loss by the sudden death on March 2, of Robert Dennis Beatson Hird, at the age of 67.

He received his medical education at Birmingham University, graduating M.B.,Ch.B., in 1905 and proceeding to M.D., a year later. He obtained the F.R.C.S. (Ed.) in 1909.

Practically the whole of his professional life was spent in his native city of Birmingham, where much of his time was devoted to public work. Among the appointments which he held at various times were: Honorary Surgeon to the Birmingham and Midland Eye Hospital, Honorary Ophthalmic Surgeon to the General Hospital—later amalgamated with the Queen Elizabeth Hospital to form the Birmingham United Hospital, and Ophthalmic Specialist to the Birmingham Education Authority. During the war he served as Midland Regional Adviser in Ophthalmology to the Ministry of Health. He was Lecturer in Ophthalmology to the University of Birmingham until 1945.

Forty years ago he was one of the band of younger men who wrote abstracts for the Ophthalmic Review under the Editorship of E. E. Henderson, and he contributed original articles occasionally to its pages. He also wrote papers in the medical journals and entered into discussions at the Annual Congresses of the Ophthalmological Society, but his literary output was not prolific and it was as a clinician that he was best known.

To all his work Beatson Hird brought those qualities which make for success. To abounding energy were added a deep knowledge and love of his speciality. He had an infinite capacity for taking pains and combined keen observation with attention to every detail. Nothing was too trivial to be noted and no effort too great which would enable an accurate diagnosis to be reached. These methods served as an example to many men now successfully practising ophthalmology in all parts of the Empire. To him ophthalmology was not confined to narrow limits, but was only part of medicine as a whole. The posts he filled on the Staffs of large General Hospitals

Lafayette

R. D. BEATSON HIRD

enabled him to exercise to the full his skill in Medical Ophthalmology and his opinion, frequently sought by his colleagues engaged in the wider fields of General Medicine and Surgery, was often of the utmost assistance in difficult cases.

He was a successful teacher. Having an easy and lucid style he was able to leave a clear impression of his subject in the mind of the student.

Throughout the years his enthusiasm remained undiminished. He was ever ready to try new methods and to adopt those which he deemed in any way an improvement on the old.

His services as Midland Regional Adviser in Ophthalmology cannot be over-estimated and it was only his strong sense of duty in those difficult days which compelled him to continue such arduous work following a severe heart attack in the early days of the war.

Outside his profession he was a keen golfer until illness prevented strenuous exercise. He was also interested in astronomy, fond of literature and a discerning philatelist. His collection of rare stamps was amongst the finest and most valuable in the Midlands.

An outstanding personality, Beatson Hird was outspoken and direct, but these qualities were always combined with courtesy and an obvious sincerity. He will long be remembered with respect by friends and colleagues alike and their sympathy is extended to his widow and family.

NOTES

Deaths

THE deaths are announced of Mr. Secker Walker, for many years of Leeds, and also of Lieut.-Col. Henry Smith, C.I.E., I.M.S., of Jullundur and Amritsar fame. Mr. Secker Walker was 82 and Lieut.-Col. Smith 91. We hope to publish notices of each in a later number.

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Award

THE Treacher Collins prize has been awarded by the Council of the Ophthalmological Society of the United Kingdom to Dr. H. S. Stannus, for his essay on nutritional eye disease.

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University of Glasgow Department of Ophthalmology Spring, 1948

DURING the month of May a series of meetings will be held in the Department on Wednesdays at 8 p.m. The General Arrangements will be similar to the series held last year. Tea will be served after the paper and a discussion will follow. The meetings

will be open to all medical practitioners and senior students interested in Ophthalmology.

May 5—Dr. I. C. Michaelson, "The Growth of Ocular Vessels"; May 12—Dr. Jas. Hill, "The Disturbances of Lacrimal Apparatus"; May 17—Dr. W. O. G. Taylor, "Wounds of the Cornea"; May 26—Mr. O. M. Duthie, "Cataract Extraction."

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Ophthalmological Society of Australia

THIS Society proposes to hold its Eighth Annual General and Scientific Meeting at Perth, Western Australia, on August 15-21, 1948, in conjunction with the Australasian Medical Congress (British Medical Association) Sixth Session.

The organisers of the Meeting are :—Dr. Claude Morlet (President of the above Society), St. George's Terrace, Perth, W.A.; Dr. Bruce Hamilton (President of the Section of Ophthalmology), 174, Macquarie Street, Hobart, Tasmania; Dr. John L. Day (Honorary Secretary of the Section of Ophthalmology), 179, St. George's Terrace, Perth, W.A.

The Society would be honoured to receive representatives of the Section of Ophthalmology, British Medical Association, and would extend to them a most cordial welcome. Intending visitors, however, should contact the Congress Office, 230, St. George's Terrace, Perth, W.A.

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Transactions of the Ophthalmological Society

THE Society holds no volumes, or only a limited number, for the following years :—

1898; 1899; 1900; 1901; 1902; 1905; 1906; 1907; 1909; 1911; 1917; 1918; 1919; 1920; 1921; 1922; 1923; 1924; 1926; 1927; 1928; 1929; 1930; 1931; 1932; 1933; 1934; 1935; 1937 (Part I).

It is felt that there may be Members willing to dispose of volumes for these years, which the Society would be grateful to buy at the rate of £1 per volume.

Communications should be made to the Honorary Secretary, 45, Lincoln's Inn Fields, London, W.C.2.

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Medical Celebrating Week of the Hungarian Medical Trade Union

AN invitation has been received from the President and Secretary General of the Hungarian Medical Trade Union to foreign Ophthalmologists to attend their "Medical Celebrations Week" in Budapest from September 4 to 12, 1948. A preliminary and detailed programme will be published within one or two months.